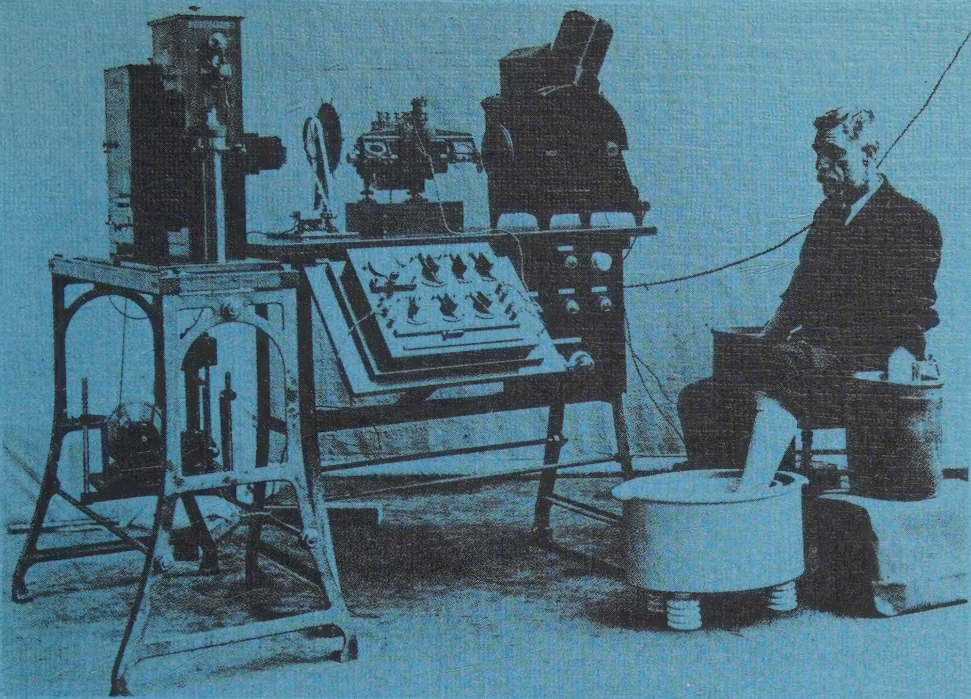




ON THE NORMAL SCALAR ECG

A NEW CLASSIFICATION SYSTEM CONSIDERING
AGE, SEX AND HEART POSITION

by

Björn Lundh

LUND 1984



From the Departments of Clinical Physiology
and Internal Medicine
University of Lund, Lund, Sweden

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Cover: Electrocardiograph from about 1908. From "Eureka - How and When the Greatest Inventions were made", 1974. Published through the courtesy of Thames and Hudson Publishers, London, England

To be published in Acta Medica Scandinavica
as Supplementum 691

Printed in Sweden
Studentlitteratur
1984

To Brigitta

Måns, Johan,
Pernilla and
Simon

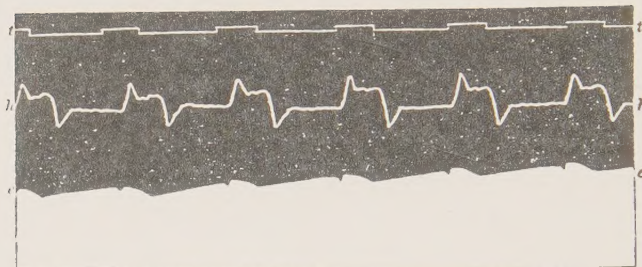


FIG. 1. Man. Heart led off to electrometer from front and back of chest (front to Hg; back to H_2SO_4).
e.e. electrometer. h.h. cardiograph. t.t. time in seconds.

"If a pair of electrodes (zinc covered by chamois leather and moistened with brine) are strapped to the front and back of the chest, and connected with a Lippmann's capillary electrometer, the mercury in the latter will be seen to move slightly but sharply at each beat of the heart."

Augustus D. Waller, M.D., 1887

The first published registration of an ECG in man (e-e) and the first published sentence on this subject.

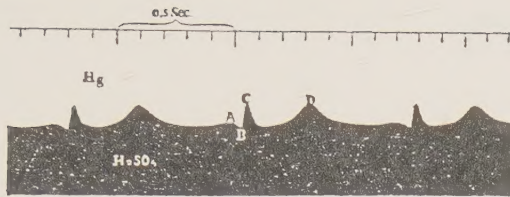


Fig. 1.

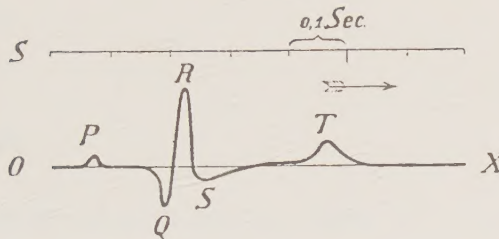


Fig. 2.

Abse. 1 mm. = 0.04 Sec. Ordin 1 mm = 10^{-4} Volt.

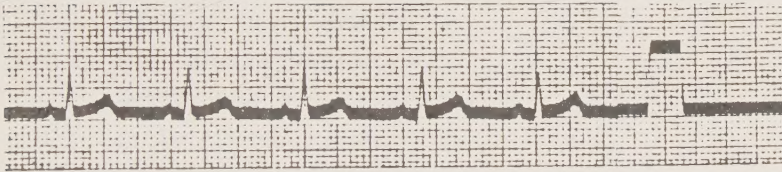


Fig 3

The different components of the ECG (Fig. 2) demonstrated by capillary electrometer (Fig. 1) and by the string galvanometer (Fig. 3) in 1903 by Willem Einthoven, who was awarded a Nobel prize in 1924 for the invention and improvement of the string galvanometer. Today, other techniques are used to record the ECG, and in the present study a modern ink jet recorder has been used.

The TRUTH is your target,
but you will find that it
has no bull's eye.

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Footnote: Peak to peak QRS-amplitudes are reported in this book only in Appendix No. 6 and PQ-, QRS- and QT-durations are reported only in Appendix No. 7.

ABBREVIATIONS AND DEFINITIONS

BP	blood pressure
BS	body surface area according to duBois et al (1916)
CC	chest circumference, measured in cm around the chest of the 4th parasternal intercostal space level
CCW	counter clockwise
CLBBB	complete left bundle branch block
CRBBB	complete right bundle branch block
CW	clockwise
DBP	diastolic blood pressure
ECG	electrocardiogram
ELAD	extreme left axis deviation = mean frontal QRS-axis $\leq -30^{\circ}$
ER	error ratio = (false positives + false negatives)/true positives
HoDST	horizontal or downward sloping ST-segments
IHD	ischemic heart disease
LAD	left axis deviation = mean frontal QRS-axis $< 0^{\circ}$
LAHB	left anterior hemiblock
LL	lower limit
LVH	left ventricular hypertrophy
MFQRS-axis	mean frontal QRS-axis
MP	mean performance = $1/2$ (sensitivity + specificity)
NS	not significant. Refers to differences and regressions where the probability for chance is greater than 0.05
OAD	obstructive airways disease
OI	obesity index according to Zilva et al (1960)
p	probability that the result is an effect of chance
Q-axis	defined through the Q-wave pattern. The direction of the Q-axis is opposite to the direction of the initial (septal) vector. The Q-axis is an alternative way to denote the electrical position of the heart (Fig. 4.3)
QI	Q-axis intermediate, e.g. 0° - 90°

QL	Q-axis to the left, e.g. less than 0°
QR	Q-axis to the right, e.g. more than 90°
SBP	systolic blood pressure
SD	standard deviation
SEM	standard error of the mean
Sensitivity	$100 \times \frac{\text{true positives}}{\text{true positives} + \text{false negatives}}$
Specificity	$100 \times \frac{\text{true negatives}}{\text{false positives} + \text{true negatives}}$
TZ	transitional zone in precordial leads
UL	upper limit
VAT	ventricular activation time
VCG	vectorcardiogram
WPW-syndrome	Wolff-Parkinson-White-syndrome

INTRODUCTION

"The method of electrocardiography is still a young plant. We may reasonably expect that it will continue to bear good fruit." With these words Willem Einthoven closed a lecture on the human electrocardiogram before the Chelsea Clinical Society in 1912. His expectations have come true. Clinical electrocardiography is now a vital 80-year old tree with many branches. Different lead systems have been developed through the years, and lately computers have been used, not only for measurements but also for interpretation. The ECG is still the most sensitive method for detecting some heart diseases, in spite of newcomers like echocardiography and various isotope techniques. The great sensitivity of the ECG is, however, not only beneficial. Minor ECG changes in the early stages of heart diseases are difficult to evaluate, as no other method is available for verification. Such minor changes may also represent normal variations. If they are labeled abnormal in a healthy subject, it may cause unnecessary harm. On the other hand, ignoring ECG-changes in a patient with early ischemic heart disease (IHD) may prevent proper handling, such as risk factor modification. To separate normal from abnormal findings is thus a frequent problem even 80 years after the birth of clinical electrocardiography.

What is normal? Is it the "ideal" or is it the "most common"?

The paradigm of normality as being something ideal is dated back to Plato (Smith 1947). Normality in this sense implies that disease is abnormal and only health is accepted as a normal state. To obtain normal values of the ECG, one therefore has to study a group of subjects free of overt as well as subclinical disease. Such a group is difficult to find, although young subjects around the age of 20 years may reasonably well fulfil such criteria of health for grown-ups. In other words, normal values are those found in a population of 20 year old "healthy" subjects. The drawback with such a definition is obvious. Many ECGs in older subjects will be classified as abnormal, thereby limiting the practical value of the ECG.

The concept of normality as being the "most common" on the other hand, implies that features are normal if they are present in a majority (say 95 %) of the subjects in a population. In reality this means that subclinical disease is accepted as normal in older ages. The prevalence of IHD, for example, increases with age, especially in men. Other factors, such as body build and arterial blood pressure, contribute to age variation. Measurements on the ECGs obtained in older ages will therefore inevitably be influenced by IHD, body build and blood pressure. Unsuccessful efforts have been made to differentiate changes due to "normal aging" from those caused by latent coronary disease (Blackburn et al 1967). "Normality" is thus a complex matter. Normal subjects may easily be classified as abnormal. This is the background for the statement that "most people do not realize what risks they run when they submit to an electrocardiogram" (Marriott 1960).

The problems concerned with the definition of the concept "normality" have been elucidated and reviewed by Simonson (1966). Normality is not solely based on statistical terms. In fact, from a purely statistical point of view, the "normal" ECG would probably be very rare. The present study is concerned with about 90 items in the ECG. Let us assume that they vary independently of each other (which of course is not true). Then, only 1 %

of the ECGs are expected to be within the 95 % confidential interval for all the 90 items. In other words, if rarity is applied as a criterion of abnormality, the normal ECG as described above would be highly abnormal. In this study of 357 healthy men and women we would expect to find 3-4 such ECGs. To find those ECGs would satisfy our curiosity, but it would serve no other purpose. The individual ECG has a clinical value only in context with the patient and it does not become normal or abnormal until it reaches the hands of the physician in charge of the patient. Prior to that, the ECG is only a set of values, some of which may fall outside the 95 % confidence limits. The physician selects only a few items, which are relevant for the situation of the patient, and then he makes a clinical diagnosis. To do so, however, he must rely on some kind of definition of "normality". Such a definition is admittedly expressed in statistical terms (for instance the 2.5 % and 97.5 % percentiles), but the final decision of normal or abnormal ECG cannot be done by anyone else but the physician in charge. He may accept a value outside the normal limits as normal for the individual patient, as well as consider a value as abnormal, although it is within the conventional statistical limits.

For practical purposes, normal limits can be delineated from a population comprising subjects, who do not show any sign of disease in applied clinical tests. Problems may be encountered, however, if less specific methods are used to select subjects for a study, in which a more sensitive method is to be analysed. As mentioned, ECG is more sensitive in diagnosing certain heart diseases than most other tests. Therefore, one could expect to find some individuals with silent heart disease, regardless of how carefully the screening is done.

In the judgement of whether a measure from an ECG is normal one should consider factors which in health influence the size and appearance of the feature measured. The most important factors are sex, age and heart position. A breakdown of the population into such subgroups will permit the individual ECG to be compared with a subsample most representative for the particular individual. Normal standards designed in the early days of ECG did not consider variations with age and heart position and only very few considered the sex differences (Kossman 1953). Many old standards used young men as reference. It was not until Simonsson published his book "Differentiation between normal and abnormal in electrocardiography" in 1961, that full consideration was given, not only to sex and age but also to heart position and body build. Unfortunately, however, subgrouping was far too extensive to be of any real value in clinical work. Such problems explain why ECGs are routinely interpreted from suboptimal standards.

Attempts to make interpretation of the ECG more uniform were made with the introduction of the so called Minnesota code (Blackburn et al 1960). This was a major event in the history of electrocardiography. It was revised during the next years until it appeared in a final edition in 1968 (Rose and Blackburn). The Minnesota code is a system, whereby the presence of various features in the ECG can be classified. Such a classification system facilitates comparison between different ECG studies by creating "a common international language" for interpreters of ECG. The Minnesota code has attained wide acceptance and undoubtedly been of crucial value in epidemiological ECG-studies.

The Minnesota code was developed for studies of men aged 40-60 years, but has also been used for women and younger men. This is probably the reason why it has been regarded as being either unspecific or insensitive.

Although it is true that the presence of some codable items in an ECG are indicative of heart disease, it is not true for other items. Such items are, for example, the high amplitude R-waves, which have been regarded as a sign of left ventricular hypertrophy (LVH), although they have been repeatedly shown to occur in young healthy men. Many problems have been due to uncritical use of the code.

The Minnesota code is designed for epidemiological studies. Some minor ECG-changes were not given codes, since they would be too time-consuming to measure and would have low specificity. The Minnesota code ST-segment items, for instance, are too coarse to include minor changes, which are of value when the early stages of IHD are to be evaluated (Evans 1952, Åstrand et al 1965). To compensate for this, a special classification system for the ST-segment was developed in Finland by Punsar et al (1968). The Punsar code is an extension and modification of the Minnesota code on the ST-segment. However, there are still many unsolved problems concerning the minor ST-segment changes. For instance, it is known that the form of the ST-segment is not the same in men and women, but the exact nature of this sex difference is not known. In this context, the Punsar code is an essential tool.

Computer-assisted interpretation of the ECG is becoming more and more common. Normal values and measurements of factors, influencing the ECG, can easily be included in a computer program and thereby increase sensitivity and specificity of the ECG. Some available programs interpret the ECG almost as accurately as trained cardiologists (Murray et al 1976, Swartz et al 1982). However, the final diagnosis of the ECG can never be left to the computer: it is always the responsibility of the doctor. Furthermore, computer-assisted interpretation will not be universally available for a good number of years. Hence, an ECG-standard for clinical use is still needed.

AIMS OF THE PRESENT STUDY

1. To study the influence of sex, age, heart position, body build and blood pressure on the form and magnitude of the ventricular ECG-deflections.
2. To provide an ECG-standard, useful for clinical work.
3. To validate a computer program for ST-segment coding, used in this laboratory.

1 MATERIAL

1.1 SELECTION OF SUBJECTS FOR THE STUDY

Participants in the study were selected at random from the computer-based civil registry in a defined urban population. The instructions were as follows:

- a. List every fiftieth man and woman born from 1915 through 1953.
- b. The person must be registered as resident in the city of Lund in one of the following four parishes: Lund's Cathedral, St Peter's Cloister, Stora Råby or All Saint's parishes.

The names, dates of birth, addresses, marital status and nationalities of 593 persons (305 women and 288 men) were received from the civil registry. The population sample reflected the population pyramid in the age group 21 to 61 years. When the study started, 21 women and 35 men had moved. Thus remained 537 subjects (284 women and 253 men) living in the parishes mentioned. These 537 subjects constituted the total material (Table 1.1).

The subjects were examined in the summers of 1975 (about 1/3 of the material) and 1976 (about 2/3).

Each participant was sent a letter containing a short description of ECG and its clinical importance, as well as the technique for recording it. The purpose of the investigation was explained. Economical compensation was not offered.

A few days after receiving the letter, the participants were contacted and booked for the examination by telephone. A short written confirmation of the appointment agreed upon was sent to the participant, together with instructions to avoid physical exercise and smoking about half-an-hour before the investigation. Those participants who did not answer the telephone-call were sent another letter together with a copy of the first letter. They were called again a few days later, and if reached, were booked for investigation. Participants not accessible by telephone were visited by the author or a laboratory technician. Persons who were hesitant to participate were subjected to slight persuasion, but not those obviously reluctant.

Table 1.1. The number of subjects selected for the study and the number of subjects finally examined. The subjects living in Lund at the time when the study started constitute the total material.

Age group	MEN			WOMEN		
	Computer-listed	Living in Lund	Examined	Computer-listed	Living in Lund	Examined
21-30 years	119	99	84	122	108	99
31-44 years	92	79	70	94	91	84
45-60 years	77	75	63	89	85	72
Sum	288	253	217	305	284	255

1.2 SUBJECTS NOT EXAMINED

The main purpose of the present investigation was to obtain normal values for certain electrocardiographic measurements. The list of subjects was therefore first compared with the patient records at the Department of Clinical Physiology of the University hospital in Lund. This department is responsible for the recording and interpretation of most of the ECGs taken in the hospital. Fourteen of the individuals were found in the records of the department. These subjects had suspected or certain disease affecting the heart and/or the lung function. These fourteen persons (eight men and six women) were therefore never called for investigation. Their mean age was 50 years (ranging from 38 to 60), and the reasons for not calling them are listed in Table 1.II.

Apart from the above-named fourteen subjects not called for examination, a further 51 subjects were not examined. Most of them (27 subjects, i.e. 5.0 %) were unwilling to participate. Five males (0.9 %) could not come for examination because of their work. Eight subjects (1.5 %) failed to appear at the time agreed on. Finally, 11 subjects (2.0 %) could not be traced. Fig. 1.1 shows the age and sex distribution of the total material, and of those subjects who were not examined.

A total of 472 subjects were thus examined, i.e. 88 % of the total sample, or 90 % of those without known disease.

Table 1.II. Age and diagnosis of the 14 subjects who were not called for investigation because of medical reasons.

Age	Diagnosis
Women	
60	Ischemic heart disease
55	Suspected atrial septal defect, heart enlarged
51	Suspected sarcoidosis
50	Systemic lupus erythematosus, chest pain
47	Suspected congenital heart disease
44	Ischemic heart disease
Men	
58	Heart disease; admitted to the cardiol. ward
56	Diabetes, abnormal exercise test
52	Ischemic heart disease
49	Ischemic heart disease
48	Anchylosing spondylitis, lung disease
47	Ischemic heart disease
45	Myocarditis 1956
48	Murmur, suspected left-to-right shunt

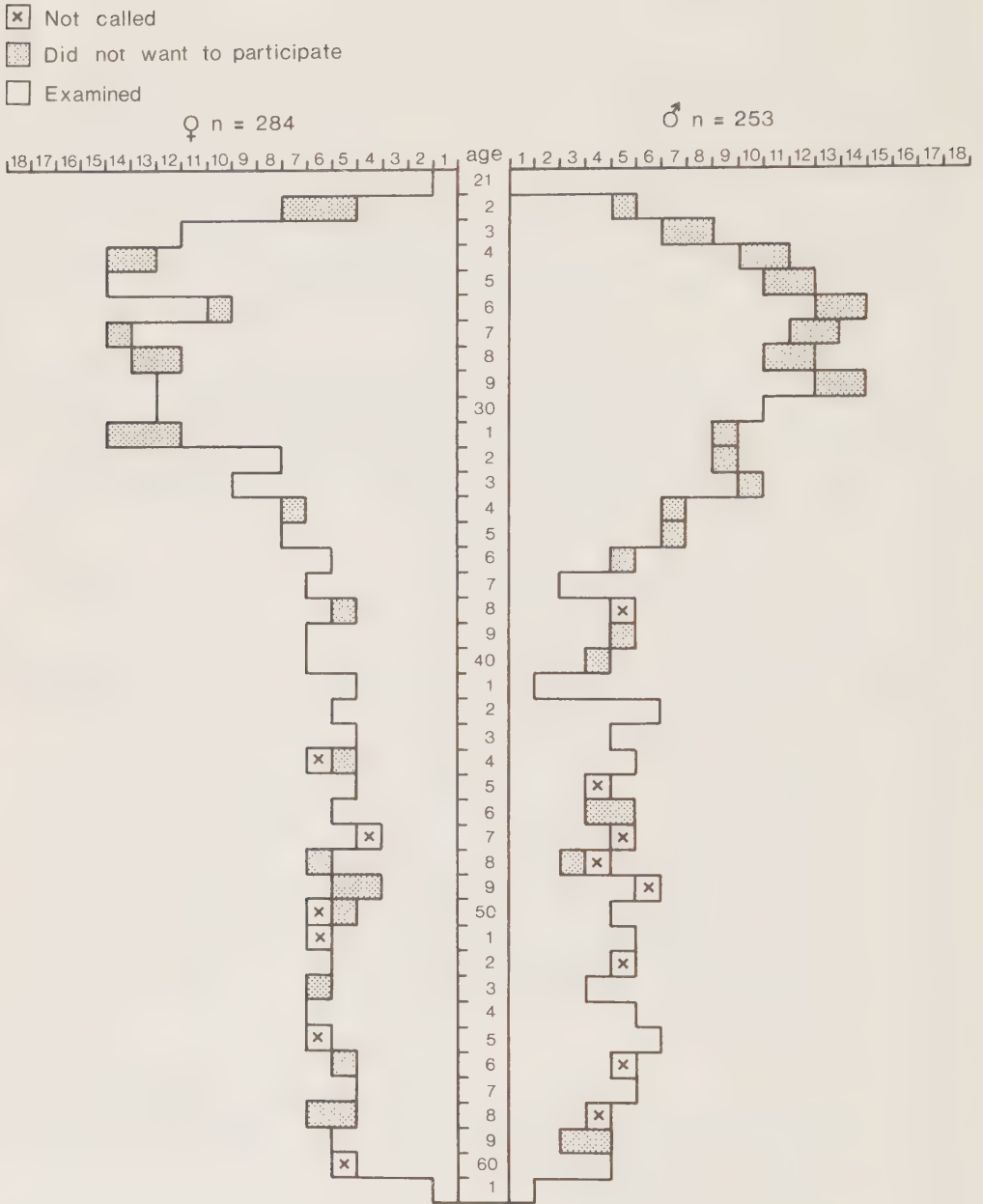


Fig. 1.1. Sex and age distribution of the total material. Those who were not called or did not want to participate are noted.

1.3 PLAN OF THE EXAMINATION

When the participant arrived for examination, the following standardized plan was rigidly followed:

1. Height and weight were recorded by a laboratory technician
2. A standard 12 lead scalar ECG and a Frank lead VCG were recorded in the supine position
3. Blood pressure in the right arm was measured by the technician
4. The subject was interviewed by the author
5. Physical examination was performed by the author

The whole procedure required about 30 minutes.

1.3.1 Interview

The interview was standardized as to number and type of basic questions asked. Effort was made to put the questions in the same manner to all of the subjects.

The interview consisted of five basic questions. The answers to these questions were classified into well-defined alternatives in advance, in order to facilitate the computer-assisted analysis (see Table 1.III). Questions I-IV were used to define subjects that might possibly suffer from diseases or other conditions that could influence the ECG-values. Question V concerned the smoking-habits of the subjects.

If the subject answered "yes" to any of the basic questions, further questioning was done in a non-standardized way. Similarly, other diseases than those mentioned in questions I-IV were asked for in a non-standardized manner. The subjects were also asked if they took drugs, or had taken drugs regularly.

1.3.2 Physical examination

Height (in cm) and weight (kg) were measured according to WHO (Rose and Blackburn 1968) with the subject undressed to the waist and without shoes. Chest circumference was measured at the level of the 4th intercostal space at the sternum, and at mid-expiration.

Blood pressure was always measured in the right arm using a cuff 13 cm wide and 34 cm long, and a mercury manometer. Systolic blood pressure was measured at the beginning of the Korotkoff sound and diastolic blood pressure was taken at the disappearance of these sounds (phase V). Blood pressure was always measured with the subject in the supine position.

The physical examination included estimation of the general condition. On inspection, special attention was paid to signs of heart, lung or thyroid disease. Auscultation of the heart was done with the participant in the supine position, while auscultation and percussion of the lungs and palpitation of the thyroid gland was done while the subject was sitting.

Heart rate was measured by a computer, from the ECG-record. This implies that it was measured after about five minutes' resting in the supine position.

Table 1.III. Questionnaire - basic questions.

I	Have you ever had sensations from the heart?		
Scoring	No	= 1	☐
	Palpitations	= 2	
	Pain	= 3	
	Other sensations	= 4	
II	Have any of your relatives suffered from heart disease?		
Scoring	No	= 1	☐
	Ischemic heart disease	= 2	
	Other heart disease	= 3	
III	Have you ever coughed daily for more than one month?		
Scoring	No	= 1	☐
	Yes	= 2	
IV	Do you suffer from or have you suffered from high blood pressure, diabetes or asthma?		
Classification	No	= 1	☐
	High blood pressure	= 2	
	Diabetes	= 3	
	Asthma	= 4	
V	Do you smoke, and if so, how much?		
Classification	Never smoked	= 1	☐
	Stopped smoking more than six months ago	= 2	
	Smokes less than 15 cigarettes a day	= 3	
	Smokes more than 15 cigarettes a day	= 4	

1.4 REASONS FOR EXCLUSION OF SUBJECTS

Subjects in a study of normal ECG-values should not suffer from abnormal conditions that might affect the heart and the ECG. The heart can be involved, either primarily as in heart disease, or secondarily to high blood pressure, lung disease, diabetes, sarcoidosis or other systemic diseases. The initial interview and examination of 472 participants aimed at finding those subjects who might possibly have conditions predisposing for ECG-abnormalities.

It is sometimes difficult to exclude heart disease even with sophisticated invasive laboratory methods. To exclude heart disease and other conditions that might possibly affect the ECG with non-invasive resources is obviously impossible. A more realistic aim is to define subjects without any clinical symptoms or signs of such conditions. In order to secure - as far as possible - a selection of subjects free from cardiac abnormalities it was decided to avoid subjects in which the history and/or physical examination gave even minor suspicion of heart disease.

1.4.1 Symptoms of heart disease

Symptoms from the heart were revealed in the interview by the standardized question: "Have you ever had sensations from the heart?".

If the answer was "No", there was no further questioning.

If, on the other hand, the answer was "Yes", or "I don't know", or "What do you mean" etc., further non-standardized questioning was done. The nature of the symptom, the time at which it occurred, its duration, frequency and effects on effort and breathing were noted. The symptom was then classified by the author into one of the three groups:

- a) Chest pain
- b) Palpitations
- c) Sensations from the chest other than chest pain or palpitations, such as feeling of heaviness, pressure, tightness etc.

The distribution of these symptoms did not show any relation to age and sex.

a) Chest pain had been experienced by seventeen individuals (9 men and 8 women). It had in all cases been severe enough to make the participant see a doctor, or seriously consider it. In most instances the pain had been felt only once or twice, usually within the last three years. One person, however, reported an episode ten years prior to the interview. It was concluded that this experience must have had great significance for the subject, having been kept in memory for such a long time.

The duration of the pain episodes ranged from ten minutes to several days. Recurrent, effort-related chest pain had been experienced only by one man.

All seventeen subjects with chest pain were excluded from the ECG-study.

b) Palpitations were recorded by 57 individuals (18 men and 39 women). This symptom was defined as an awareness of strong heart beats without relation to exercise or emotional stress. No consideration was taken to

regularity of the beats. Duration of the attacks ranged from a few seconds to several minutes, except in two men with paroxysmal tachycardia, in whom the attacks sometimes lasted for several hours.

Palpitations were more common in women (15 %) than in men (8 %). The difference is significant ($\chi^2 = 4.8$, $p < 0.05$). The subjects reporting palpitations and those lacking this symptom were compared with respect to a number of factors.

There was no significant relationship between palpitations and heart rate at rest, weight, arterial blood pressure, smoking habits or thyroid enlargement. There were slight differences in age and height between subjects with and without palpitations.

Palpitations in women over thirty years of age were, however, related not only to height, but also to weight and heart rate. Lean and short women with a high heart rate at rest thus reported palpitations more frequently in this age group (Table 1.IV).

It was felt that these observations did not suggest an association between palpitations and heart disease. Therefore fifty-five of the participants with palpitations were included in the ECG-study. Two men with paroxysmal tachycardia were excluded, as the diagnosis had earlier been verified by physicians. One of these men had a WPW-syndrome.

c) Sensations other than chest pain or palpitations were reported by 33 subjects (17 men and 16 women). These sensations were described as feelings of "heaviness", "pressure" or "tightness" and lasted up to 10 minutes. Two of the women reported that their symptoms were associated with effort. Most of the subjects had only had one or two such episodes during the last two or three years. All 33 subjects were nevertheless excluded from the ECG-study, since the symptoms might be a sign of heart disease.

Table 1.IV. Height, weight and heart rate at rest in women over 30 years of age reporting palpitations compared with the other women (31-60 years).

	Palpitation n=30	No palpitation n=109	Significance
Height, M $\pm$ SD	162 $\pm$ 5.4	165 $\pm$ 4.9	**
Weight, M $\pm$ SD	58.1 $\pm$ 9.5	63.1 $\pm$ 10.3	*
Heart rate, M $\pm$ SD	82 $\pm$ 13.4	75 $\pm$ 13.9	*

1.4.2 Signs of heart disease

Signs of heart disease at auscultation were classified in one of the following categories:

- a) Abnormal heart sounds
- b) Significant murmurs
- c) Physiological murmurs

The distribution of these findings did not show any relation to age and sex.

a) Abnormal heart sounds were found in 3 % of the examined subjects (4 men and 10 women). There was no statistically significant relation to age or sex. A second sound, that was split in expiration as well as inspiration, was found in five subjects. A fourth sound was heard in four subjects and a systolic click in two. A split first sound was heard in one, and a third sound in another subject. The fourteenth person had a significant murmur as well as a third sound.

Abnormal heart sounds are often found with abnormal haemodynamics or low compliance of the walls of the left ventricle. Systolic clicks may i.a. be an early sign of cardiomyopathy. All fourteen subjects with abnormal heart sounds were consequently excluded from the ECG-study.

b) Significant murmurs were present in 2 % of the individuals (5 men and 6 women). These murmurs were all systolic and were suspected of indicating mitral incompetence in four subjects (1 man, 3 women), aortic stenosis in two men, subvalvular aortic stenosis in one man and posterior leaflet syndrome in one woman. The murmur was of high frequency and short duration in one man, but did not fulfill the criteria for a physiological murmur (see below) because of late onset. The other two women in this group had murmurs of high intensity, heard over the entire precordial area.

None of the subjects had any apparent symptoms of heart disease and all of them had normal chest radiography.

All of these eleven participants with significant heart murmurs, were, however, excluded from the ECG-study.

c) A physiological murmur was defined as a systolic murmur of high frequency and low, often decreasing intensity (grade 1-2, according to Levine), starting immediately after a normal first sound. The murmur was not pansystolic. It was best heard along the left sternal border and often also at the apex.

Physiological murmurs were more common in women ($73/255 = 29\%$) than in men ($40/217 = 18\%$), which is a significant difference ($\chi^2 = 6.14$, $p < 0.02$). Men with murmurs were younger ($p < 0.02$) than the other male participants. Women with murmurs had higher systolic blood pressure. Pulse pressure was higher in subjects with murmurs (Table 1.V).

Subjects with and without physiological murmurs did not differ as regards height, weight, heart rate, symptoms of heart or lung disorders, hereditary disposition for heart disease or smoking habits.

The association of higher systolic blood pressure and pulse amplitude with the presence of a physiological murmur may be explained by a higher activity of the sympathetic-adrenal system.

Table 1.V. Age (years), systolic, diastolic and pulse pressure (mm Hg) and heart rate in subjects with and without insignificant murmur. Significance of differences have been evaluated with Student's *t*-test.

* = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$, NS = not significant.

		MEN			WOMEN		
		Physio- logical murmur n=40	No murmur n=168	Signi- ficance	Physio- logical murmur n=73	No murmur n=170	Signi- ficance
Age	M	33.9	38.6	*	37.6	36.4	NS
	SD	9.7	11.7		11.9	11.1	
Systolic blood pressure	M	132.4	128.0	NS	126.1	120.6	**
	SD	16.4	13.0		16.8	13.0	
Diastolic blood pressure	M	80.1	80.2	NS	78.6	77.7	NS
	SD	12.0	9.2		10.6	8.0	
Pulse pressure	M	52.3	47.8	*	47.5	42.9	***
	SD	13.0	10.2		9.9	9.4	
Heart rate	M	75.4	72.4	NS	75.1	77.2	NS
	SD	14.8	15.0		14.2	12.9	

1.4.3 High blood pressure

Criteria for abnormally high arterial blood pressure vary with i.a. measurement conditions. In the present study it was decided to define hypertension as a systolic (SBP) or a diastolic (DBP) blood pressure above the upper limit of the 95 % confidence interval, as determined from regression analysis of the present material (Fig. 1.2 and 1.3).

The results of the regression analyses were:

MEN: SBP = $123 + 0.17 \times \text{AGE}$, SD = 13.5; $r = 0.14$, $p = 0.03$
 DBP = $70 + 0.28 \times \text{AGE}$, SD = 9.2; $r = 0.33$, $p < 0.001$

WOMEN: SBP = $103 + 0.54 \times \text{AGE}$, SD = 12.8; $r = 0.43$, $p < 0.001$
 DBP = $64 + 0.37 \times \text{AGE}$, SD = 7.7; $r = 0.48$, $p < 0.001$

The distributions of systolic and diastolic blood pressures in the present study conform well with the distribution in the rural population of Dalby community, which is situated close to the city of Lund (Thulin et al 1978).

Of interest is the observation, that the diastolic blood pressure in men increases more with age than the systolic blood pressure. In other words, the pulse pressure decreased with age in men, which is in contrast to the findings by Thulin et al (1978). The reason for this discrepancy is unknown.

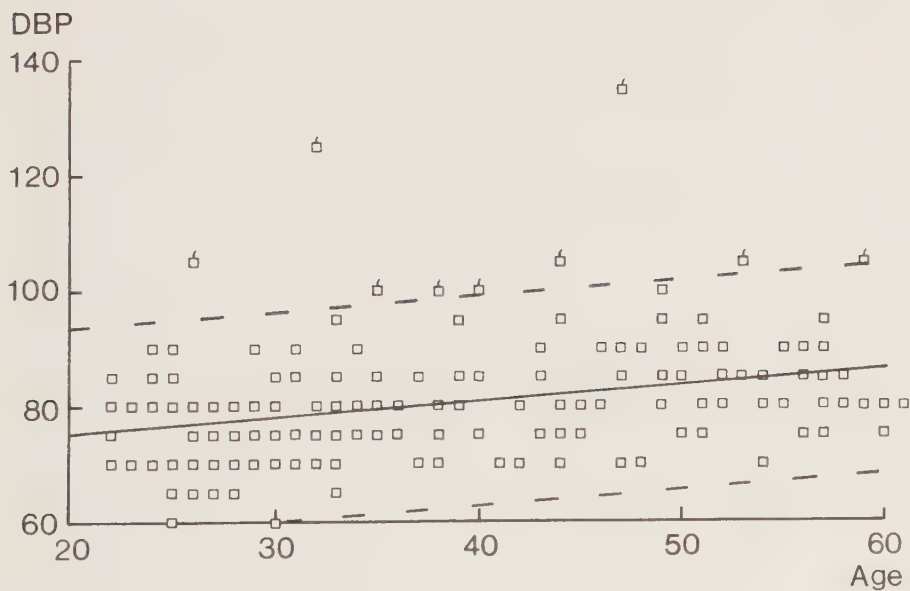
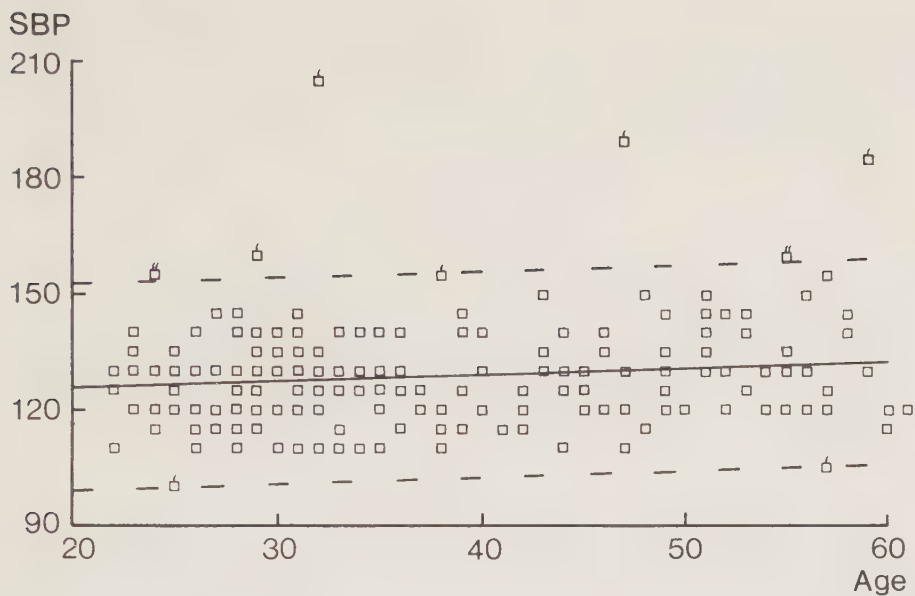


Fig. 1.2. Distribution of systolic (SBP) and diastolic (DBP) blood pressure in 217 men aged 21-60 years. The limits of the 95 % confidence interval are noted. Blood pressure in mm Hg. Overlapping data are not indicated within the 95 % confidence interval. Outside this interval each tag on the symbol represent one individual.

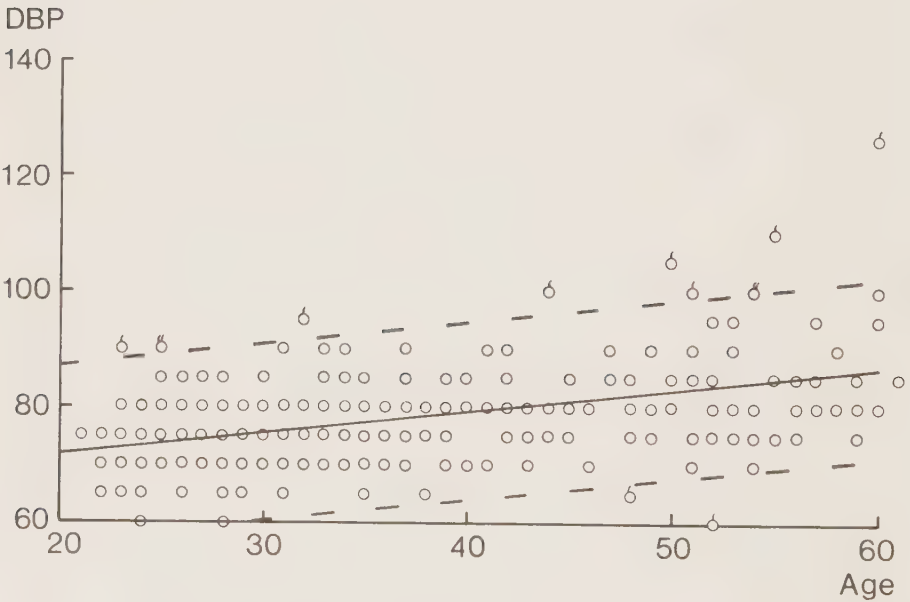
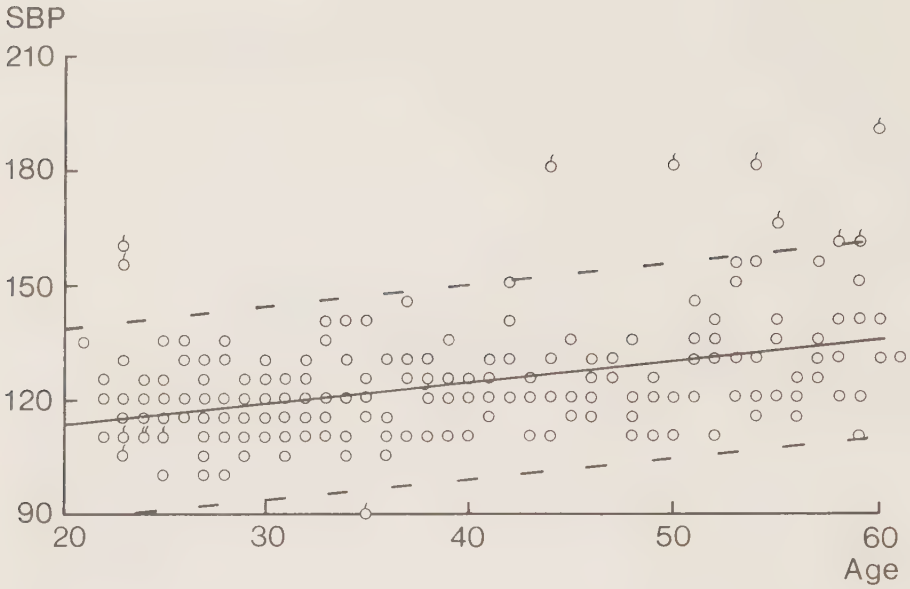


Fig. 1.3. Distribution of systolic (SBP) and diastolic (DBP) blood pressure in 255 women aged 21-60 years. The limits of the 95 % confidence interval are noted. Blood pressure in mm Hg. Overlapping data are not indicated within the 95 % confidence interval. Outside this interval each tag on the symbol represents one individual.

Twenty-four subjects (10 men and 14 women) had a systolic or diastolic blood pressure exceeding the upper limit of the 95 % confidence interval and were therefore excluded. Another three women were normotensive, but under treatment for hypertension, and they were therefore excluded also. Thus 27 individuals (5.7 %) were excluded on account of high blood pressure.

1.4.4 Obstructive airways disease (OAD)

Ten patients (2 %) were excluded from the study because of OAD. Four of them were obstructive (expiratory rhonchi) at auscultation. Another four were not obstructive at examination but were being treated for OAD. One woman had severe nocturnal coughing, which was probably due to smoking. One man had severe emphysema with cyanosis.

1.4.5 Other reasons for exclusion

The last group of excluded subjects consisted of 6 men and 5 women. They were excluded from the study either because of conditions with possible influence on the heart and significant arrhythmias or signs of abnormality in the conduction system demonstrated at ECG. The reasons for exclusion were as follows:

diabetes (2 women), sarcoidosis (1 man), alcoholism (1 man),
epilepsy (1 woman), funnel chest (1 man, 1 woman),
ventricular ectopic beats in bigeminy (1 man),
atrial ectopic beats in bigeminy (1 man),
multiple atrial ectopic beats (1 man, 1 woman)

Of the four last subjects excluded, only the man with the ventricular ectopic beats in bigeminy was aware of the arrhythmia. He could in fact provoke it voluntarily. The other three subjects (with atrial ectopic beats) had no symptoms of arrhythmia and were consequently excluded by means of ECG-criteria and auscultatory findings. Arrhythmias and especially asymptomatic arrhythmias, found in a "normal" population may of course be a normal finding. In this study, however, only occasional ectopic beats have been regarded as normal.

Another six subjects had ECG-signs of arrhythmia or abnormalities in the conduction system, but had already been excluded from the study because of symptoms and signs of heart disease or hypertension. The ECG abnormalities in these subjects consisted of right bundle branch block (1 woman), atrial fibrillation (2 women), pre-excitation (1 man) and first degree AV-block with a PQ-time of 0.29 s (1 woman) and of 0.24 s (1 man).

None of the subjects had symptoms or signs of thyroid disease, although 40 women (16 %) and 5 men (2 %) had a clearly palpable thyroid gland. Some of these subjects complained of sweating or palpitations, or both, but they all had normal thyroid function, demonstrated by analysis of blood thyroid hormones.

The age and sex distribution of the examined subjects are illustrated in Fig. 1.4. The reasons for the exclusion are concluded in Table 1.VI.

■ Not accepted

□ Accepted

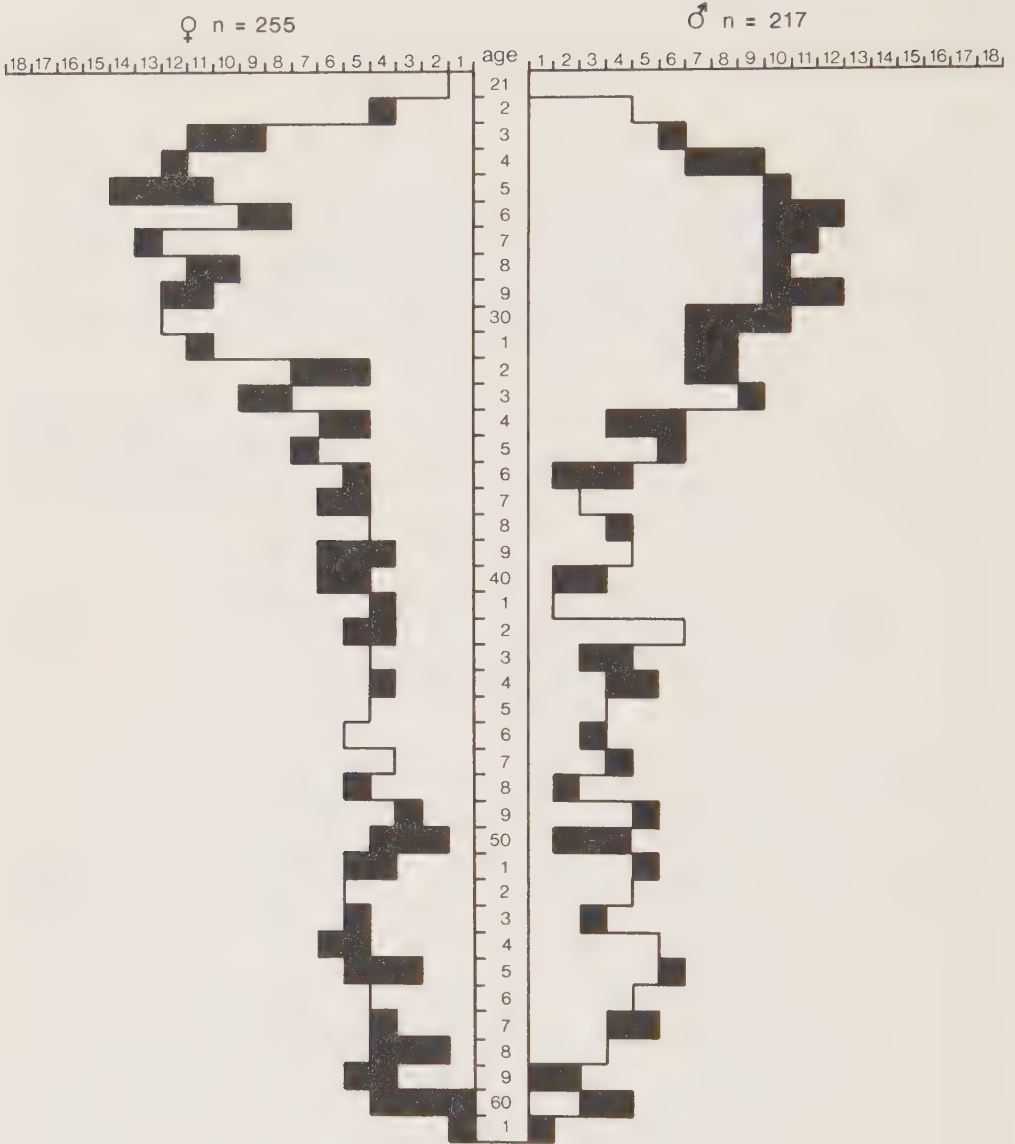


Fig. 1.4. Sex and age distribution of the examined material. Subjects who were not accepted are noted.

Table 1.VI. Number of excluded subjects and the reasons for exclusion.

Examined participants	472	
	255 women (100 %)	217 men (100 %)
Excluded because of:		
Possible heart disease	34 women (13 %)	34 men (16 %)
High blood pressure	17 women (7 %)	10 men (5 %)
Obstructive lung disease	5 women (2 %)	4 men (2 %)
Other reasons	5 women (2 %)	6 men (3 %)
Accepted for study	194 women (76 %)	163 men (75 %)

1.5 MANAGEMENT OF ABNORMAL FINDINGS

One of the motives for participation in the present study was the desire of the subjects to obtain a health-check. Therefore all subjects with definite pathological changes were referred to appropriate medical instances for further diagnosis and treatment.

More difficult to handle were subjects with borderline abnormalities, especially if they did not have symptoms. In the present study, however, it was decided to examine all subjects with abnormal heart sounds or significant murmurs with a chest radiograph. This was normal in all cases. A few of the subjects were nevertheless referred to the Department of Cardiology for investigation. As mentioned, subjects with enlarged thyroid glands were investigated with analysis of thyroid hormone level.

1.6 COMPOSITION OF THE ACCEPTED SAMPLE

The sample finally accepted to delineate normal values of the ECG consists of 357 individuals (163 men and 194 women). Age and sex distribution were shown in Fig. 1.4. All subjects were Caucasians, except one male negro from West Africa. From the names of the participants it was furthermore concluded that another 22 (6.2 %) subjects may originate from countries outside Scandinavia. Fifteen names suggested Southern European origin and seven had British or German names.

1.7 ANTHROPOMETRIC DATA IN THE ACCEPTED SAMPLE

The body surface area was calculated according to the formula of Du Bois and Du Bois, i.e. $\text{weight}^{0.425} (\text{kg}) \times \text{height}^{0.725} (\text{cm}) \times 71.84$ (Du Bois et al 1916). An obesity index (Zilva et al 1960) was calculated as $\text{weight}/\text{height}^3 (\text{kg}/\text{m}^3)$. Mean values and standard deviation of body surface, obesity index, age, height, weight and chest circumference in three age groups are reported in Table 1.VII. Analysis of the variance has been made and significant change of body build was only found for men. Older men are shorter and heavier and have a wider chest and a large obesity index. In women, the trends are the same, although they did not attain statistical significance.

Table 1.VII. Anthropometric data on the subjects in the accepted sample, subdivided into three age groups A (21-30 years), B (31-44 years) and C (45-60 years). n = number, H = height (cm), W = weight (kg), CC = chest circumference (cm), BS = body surface area (m²) and OI = obesity index. Analysis of the variance (F-test) has been performed and significant influences of age (F-values) are reported.

* = $p < 0.05$, ** = $p < 0.01$, NS = not significant.

M = mean value, SD = standard deviation, grand M = mean value of all subjects (men or women). ν_1/ν_2 = degrees of freedom

		MEN							WOMEN						
AGE group		n	Age	H	W	CC	BS	OI	n	Age	H	W	CC	BS	OI
A	n	66							83						
	M		26.4	180	71.1	94.5	1.89	12.3		26.4	166	60.2	87.5	1.66	13.3
	SD		2.3	6.4	8.4	4.8	.13	1.3		2.5	5.6	9.9	7.6	.14	1.9
B	n	51							63						
	M		36.3	178	75.2	97.3	1.93	13.4		36.3	165	61.1	87.8	1.67	13.5
	SD		4.3	6.3	8.7	5.2	.13	1.4		4.2	4.9	8.7	7.0	.12	1.9
C	n	46							48						
	M		52.5	176	74.2	98.3	1.90	13.7		51.5	164	62.1	89.9	1.68	14.0
	SD		4.2	5.4	9.0	6.9	.12	1.9		4.3	4.8	8.0	7.3	.11	1.6
GRAND		163							194						
M			36.9	178	73.3	96.4	1.90	13.0		35.6	165	61	88.2	1.67	13.5
SD			11.3	6.3	8.8	5.8	.13	1.6		10.6	5.2	9.1	7.4	.13	1.8
F-value		$\nu_1/\nu_2 = 2/160$	** 6.0	* 3.8	** 5.0	NS 1.6	*** 14.4		$\nu_1/\nu_2 = 2/191$	NS 2.4	NS 0.7	NS 1.8	NS 0.4	NS 2.3	

2 METHODS

The recording of the 12-lead electrocardiogram has been well standardized, particularly through the recommendations of the American Heart Association (1938 and 1967). The present study utilized a computer, mainly for averaging the ECG and the coding of ST-segment changes in chest leads over the left ventricle. Averaging implies a 3.3 % reduction of the QRS-amplitude with the system used in this study (Werner 1976). The use of a computer necessitates a methodological description and validation.

2.1 ECG-RECORDING

The recording of the ECG was done by a modified standard 8-channel ink jet recorder (EM61, Siemens-Elema AB), which was connected on-line to a computer (PDP 8/E, Digital Equipment Corporation) for averaging the ECG and making certain calculations. The averaging routine (Jonson et al 1976) consisted of the following steps:

- a) The ECG-signal from eight leads (six chest leads and two extremity leads, I and II) were sampled during 15 s and stored on a disc. The two extremity leads contain the information required to construct the other four standard extremity leads (see below).
- b) The computer identifies preliminary QRS-complexes, by analysis of the summed amplitude differences of three chest leads.
- c) The heart rate was determined and the ECG-complexes to be averaged were selected. In order to be included in the average, an ECG-complex must fulfil specific criteria for R-R-interval length and to some extent QRS-morphology. Beats not fulfilling these criteria were eliminated from the average (Jonson et al 1976). That this technique effectively eliminated ectopic or otherwise aberrant beats was checked by visual inspection of all of the beats averaged. Altogether 15 aberrant beats were found among the 357 subjects of the accepted sample and none of these beats were included in the average.
- d) The selected ECG-complexes were averaged.
- e) The computer constructs the four non-recorded extremity leads, using the formula given by Goldberger (1953) among others. The ECG is displayed on the jet recorder at the paper speed of 50 mm per s. A standard calibration of 1 mV = 10 mm deflection is also presented (Fig. 2.1). If arrhythmia was present, a non-averaged continuous record was also obtained.
- f) The computer presents ST-codings according to Punsar et al (1968) in leads V₄, V₅ and V₆ (Werner et al 1976).

The standard 12-lead ECG obtained for the present study consists of the six extremity leads aVL, I, -aVR, II, aVF, III and the six unipolar chest leads V₁-V₆. These were recorded from the positions on the chest recommended by the American Heart Association (1938).

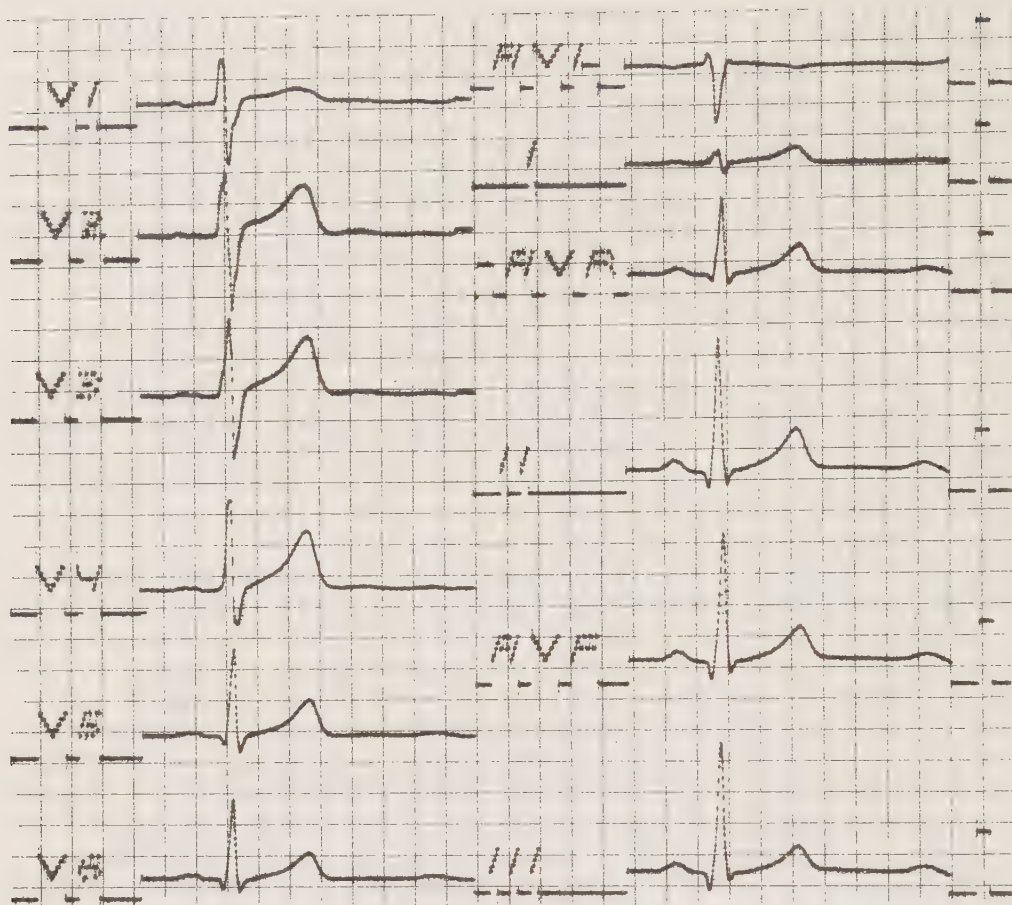


Fig. 2.1. ECG from a young man, aged 24 years. Note the vertical position, the high amplitudes and the steep upward slopes of the ST-segment. Standard calibration is seen to the right. 10 mm = 1 mV.

2.2 MEASUREMENT ON THE ECG

Visual measurements were made of the amplitudes of Q, R, S, ST-junction and T, to the nearest 0.1 mm. Duration of the Q-wave was also measured to the nearest 0.1 mm, i.e. to the nearest 2 milliseconds. A lens (magnifying six times) was used for all visual measurements. The measurements were done as shown in Fig. 2.2. The mode of measuring was generally in accordance with the recommendations of WHO (Rose and Blackburn 1968).

Measurement of ST-inclination is illustrated in Fig. 2.3. The line a-b is an extension of a line drawn to cover as much of the ST-segment as possible. The inclination was expressed in mV per second.

The visual measurements were made by three observers, well-trained for ECG-coding (a registered nurse, a laboratory technician, and a physician). The 472 ECGs were randomly distributed among the three observers, who received approximately equal numbers of ECGs for measurement. All measurements and coding were done blindly, i.e. the observers did not know the age nor the sex of the subjects. In order to find out if there were any differences between the observers, ten randomly selected ECGs were measured by all three of the observers. No statistically significant differences were encountered.

Calibration of the ECG-machine was done prior to each individual ECG. Each calibration signal was measured in the same way as all amplitude deflections, i.e. with a lens magnifying six times and to the nearest 0.1 mm. Each observed QRS-deflection amplitude (mm) was then multiplied with a factor:

$$\frac{\text{expected calibration amplitude (= 10.0 mm)}}{\text{observed calibration amplitude (mm)}}$$

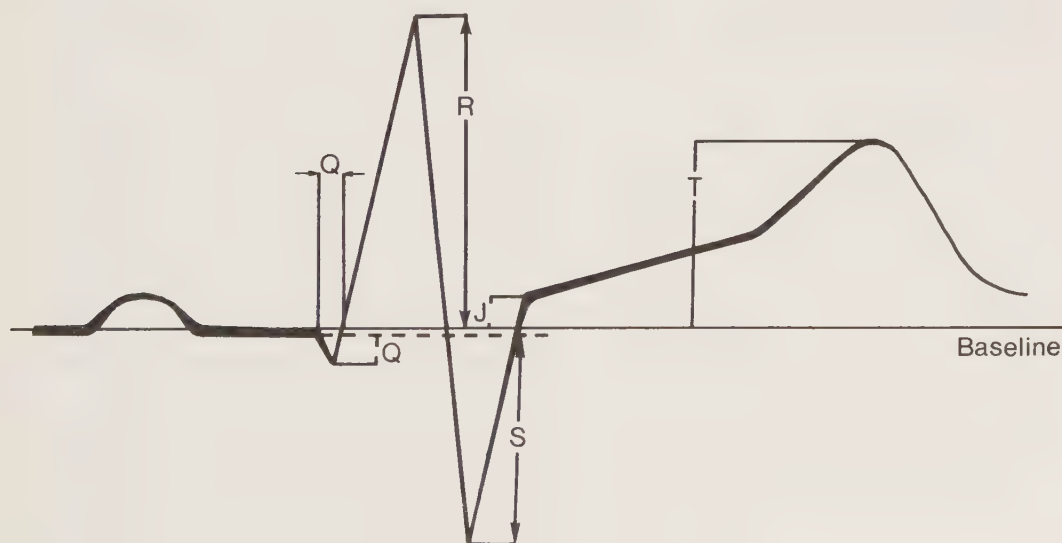


Fig. 2.2. Measurements on the ECG. The mode of measuring was in accordance with the recommendations of WHO (Rose 1968), except for the height of the T-wave, which was measured from the PQ-segment instead of the TP-segment.

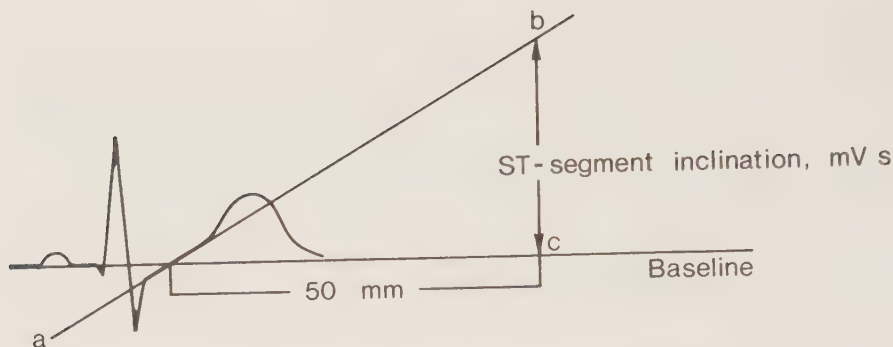


Fig. 2.3. Measurement of the ST-segment inclination. A straight line (a-b) was drawn so as to extend the apparent mean direction of the ST-segment. The inclination, expressed as mV/s, can then be calculated as the level above or below the baseline reached after one second. The paper speed used in this study is 50 mm/s.

2.3 STATISTICAL ANALYSIS

Conventional statistical methods have been used for the calculation of the arithmetical mean and its standard error and standard deviation. The significance of differences between the two mean values has been examined using Students' t-test. When three or more groups were compared, the differences between the mean values were examined according to the principle of analysis of variance, using the one-tailed F-test (Snedecor 1971).

For qualitative variables, differences in frequency were evaluated with the χ^2 -test with Yates' correction.

Correlations between variables were analysed with single or multiple regression.

The null hypothesis was rejected at the significance level of 5 % ($p < 0.05$), 1 % ($p < 0.01$) and 0.1 % ($p < 0.001$). Significance of the difference is denoted with an asterisk, * ($p < 0.05$), ** ($p < 0.01$) or *** ($p < 0.001$). When the probability for chance was greater the difference was denoted NS (not significant). However, some values for t and F are presented with their p-values even if p does not indicate statistical significance.

Normal limits have been given as percentiles, as most of the distributions in this study are skew. In samples of 39 subjects or more the upper and lower limits represent the 97.5 % and 2.5 % percentiles, respectively. In smaller samples, these percentiles cannot be defined. Therefore, in samples of 19-38 subjects the upper and lower limits represent the 95 % and 5 % percentiles, respectively. In samples of less than 19 subjects the total range is given. The percentiles have been calculated according to Snedecor (1971).

2.4 METHODOLOGICAL ASPECTS OF ST-SEGMENT CODING

A scrutiny of the literature on "minor" or "non-specific" ST-changes (i.a. Taran 1958, Averill 1960, Joy 1981) shows that emphasis is placed on the need for using a well defined classification system with rigid methodological details.

Coding of the ST-segment is based on depression of the ST-junction as well as the inclination of the ST-segment. Horizontal or downward sloping ST-segments are well classified in the Minnesota code 4.1-3 (Fig. 2.4). Ascending ST-segments, however, are less precisely classified. In the Minnesota code 4.4 ST-junction depressions of 0.1 mV or more in combination with ascending ST-segments are taken as one group without consideration to the configuration of the ST-segment. A modification of the Minnesota code was therefore proposed by Punsar et al (1958). In the modification (here called the "Punsar code") (Fig. 2.5), the ST-depressions are divided into three categories. The magnitude of the ST-segment depression is measured from the PR-segment baseline at the beginning of QRS, as recommended in the original Minnesota code. In the ischemic (I-type) category of ST-depression, the ST-segment is horizontal or downward sloping. According to the magnitude of ST-junction depression, the I-type changes are divided into five subcategories. In the second main category of ST-depression, the ST-segment is slowly ascending (S-type) and does not reach the baseline before the (apparent) beginning of the T-wave. The category is divided into four subcategories according to the magnitude of the ST-junction depression. In the third main category of ST-depression, the ST-segment is rapidly ascending (R-type) and traverses the baseline before the beginning of the T-wave. This category is divided into three subcategories according to the magnitude of the ST-junction depression. Unfortunately, there is no clear definition of the beginning of the T-wave.

ST-segments are classified as normal if ST-junction depression is less than 0.05 mV and if ST-segment traverses the baseline before the "apparent" beginning of the T-wave.

The computer program for the ST-segment classification was developed by Werner et al (1976) and added to the program for averaging ECGs (Jonson et al 1976), that is routinely used in our laboratory.

The beginning and end of the QRS-complex are calculated from leads V₂, V₄ and V₆. The sum of the absolute amplitude differences over 10 ms in these leads (SAD) was calculated in sample points of the averaged ECG, 5 ms apart. A tentative beginning of the QRS was located by searching from inside the QRS until SAD was below 0.12 mV in two of three consecutive points. A tentative end of the QRS was found in a similar manner. If the resulting QRS-width and ST-junction amplitude were reasonable, the tentative beginning and end were accepted.

The measurements used by the computer for ST-segment classification are illustrated in Fig. 2.6. The ST-segment was classified on the basis of six measures: the amplitude of the ST-junction, the amplitude and position (in time) of the lowest point in the first two thirds of ST-segment, the ST-area below the zero reference level and two ST-segments' slopes (the early and the late slope). The ST-T-segment was assumed to end $400\sqrt{60/\text{HR}}$ ms after the beginning of the QRS.

S-T junction (J) and segment depression

(Do not code in presence of ventricular conduction defects 6-4, 7-1, 7-2, 7-4.)

- 4-1 S-T-J depression 1.0 mm or more and S-T segment horizontal or downward sloping in any of leads I, II, aVL, aVF, V1, 2, 3, 4, 5, 6 (requires a T-wave code in 5).
- 4-2 S-T-J depression at least 0.5 mm and less than 1.0 mm and S-T segment horizontal or downward sloping in any of leads I, II, aVL, aVF, V1, 2, 3, 4, 5, 6. (Requires a T-wave code in 5.)
- 4-3 No S-T-J depression as much as 0.5 mm, but S-T segment downward sloping and segment or T wave nadir at least 0.5 mm below P-R baseline in any of leads I, II, aVL, V2, 3, 4, 5, 6. (Requires a T wave code in 5.)
- 4-4 S-T-J depression of 1.0 mm or more and S-T segment upward sloping, or U-shaped, in any of leads I, II, aVL, V1, 2, 3, 4, 5, 6.

Fig. 2.4. The Minnesota code 4 (ST-segment items).

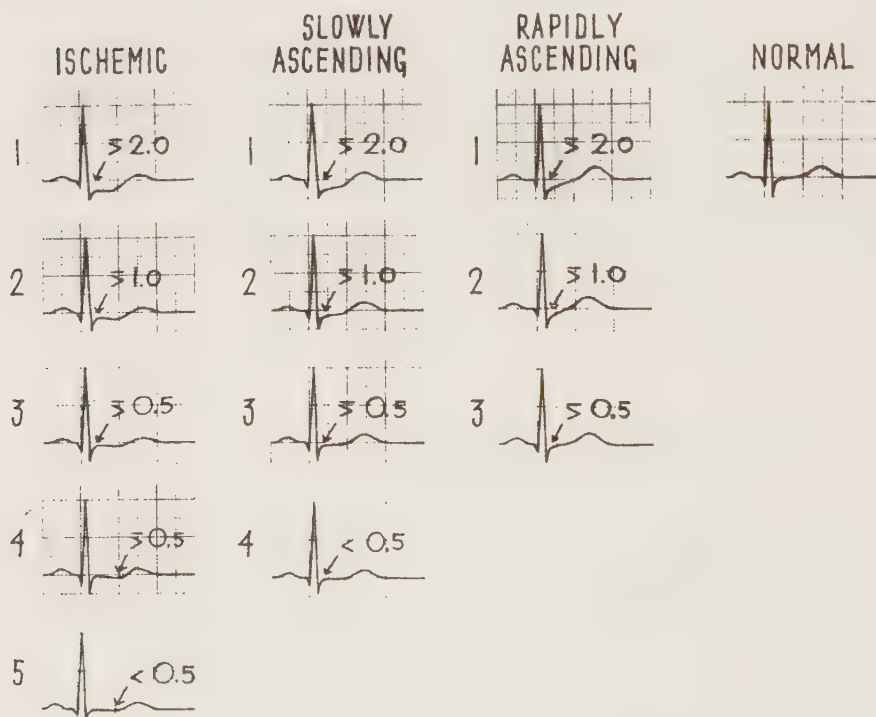


Fig. 2.5. ST-segment classification according to Punsar et al (1968). The figures represent measurements in mm in relation to the PQ-segment. 1 mm = 0.1 mV. Published through the courtesy of Dr Sven Punsar.

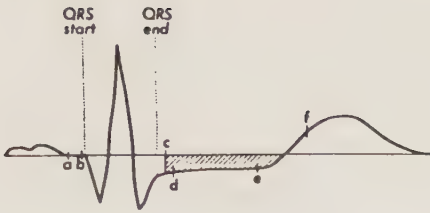


Fig. 2.6. Computer measurements on the ST-segment. Point a lies 20 ms and point b 5 ms before the QRS. The zero reference level is taken as the mean of all sample values between points a and b, end points included. Point c lies 10 ms after the QRS end. The ST-area (hatched) represents the integral of the negative ST-T-amplitudes. Points d, e and f lie 20, 120 and 180 ms after the end of the QRS when the heart rate is 60/min. These distances change in inverse proportion to the heart rate. The early slope is measured by linear regression between amplitude and time on the segment d-e, the late slope similarly on the segment e-f. The ST-J-amplitude is obtained by backwards linear extrapolation of the ST-segment through a point 15 ms after the QRS end (not indicated) onto the QRS end. The complete ST-T-analysis also requires the position (in time) and amplitude of the maximum in the ST-T, and the positions and amplitudes of the lowest point in the ST-T and the lowest point in the first 2/3 of the ST-T. The ST-T-segment is assumed to end $400 \times \sqrt{60/HR}$ ms after the start of the QRS. HR is the heart rate in beats/min. From Werner et al (1976).

	Horizontal or downward sloping	Slowly ascending	Rapidly ascending
Common criteria	$ES \leq 0$ or $LS \leq 0$; $PMIN \geq 65$	not I1-I6; $1,3 \times ES + 20 \times STJ \leq 0$	not I1-I6; not S1-S4
Specific criteria	I1: $STJ \leq -0.2$ I2: $-0.2 < STJ \leq -0.1$ I3: $-0.1 < STJ \leq -0.05$ I4: $-0.05 < STJ \leq 0$; $AMIN < -0.05$ I5: $-0.05 < STJ \leq 0$; $AMIN \geq -0.05$ I6: $0 < STJ$	S1: $STJ \leq -0.2$; $STA \geq 4$ S2: $-0.2 < STJ \leq -0.1$; $STA \geq 4$ S3: $-0.1 < STJ \leq -0.05$; $STA \geq 3$ S4: $-0.05 < STJ \leq 0$; $STA \geq 2$	R1: $STJ \leq -0.2$ R2: $-0.2 < STJ \leq -0.1$ R3: $-0.1 < STJ \leq -0.05$

Explanations:
 ES=early slope; PMIN=distance from the end of the QRS to the lowest point in the first 2/3 of the ST-T segment, ms AMIN=amplitude of that point, mV
 LS=late slope, mV/s STA=ST area, mV x ms
 STJ=ST-J amplitude, mV

Fig. 2.7. Criteria used by the computer to classify ST-segments according to Punsar (1968). From Werner et al (1976).

Criteria for the different Punsar codes were defined from these measurements (Figs. 2.6 and 2.7). The algorithm used for defining a slowly ascending ST-segment was found empirically after analysis of the ST-segments in a pilot study (O. Werner, personal communication).

The computer program was previously tested on 217 patients performing an exercise test (Werner 1976). One averaged record at rest (lead V₅) and one at 6 min of the highest work load (lead CH₅) were selected from each patient and presented to two independent observers for classification according to Punsar. It was found that computer-observer comparisons yielded no more disagreements than between-observer comparisons. At rest, there was 75 % agreement in the computer-observer comparison. The disagreements were mainly borderline (22 %). Moderate disagreement occurred in 3 % of the records. It was usually not possible to say whether the computer or the observers were right in the borderline disagreements. Borderline disagreements are illustrated in the ECG in Fig. 2.8. Real coding errors by the computer were found in about 3 % of the records.

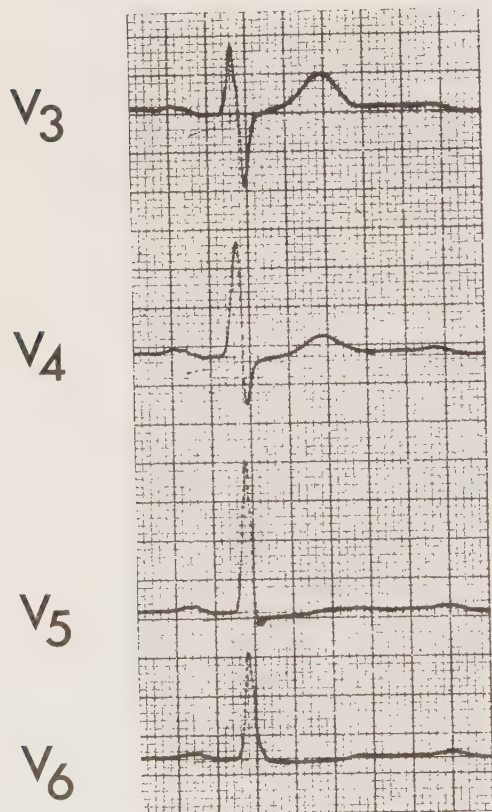


Fig. 2.8. Computer-observer disagreement in ST-segment classification. Leads V₃-V₆ in the ECG of a man, aged 52 years. The ST-segment in lead V₃ is normal. The computer coded R₃ in V₄, S₃ in V₅ and I₄ in V₆. Codes according to Punsar (1968). The two observers disagreed on V₆ and coded I₅ and S₄ respectively. Who is right?

2.5 VALIDATION OF THE COMPUTER PROGRAM

Although the computer program for ST-segment classification appears reliable and valid for clinical purposes, it was considered desirable to also verify its validity in the present material of normal subjects. Two experienced observers (a physician and a registered nurse) independently classified the ST-segments in leads V₄, V₅ and V₆ according to Punsar. They were not aware of the age nor the sex of the subjects, nor of the codes proposed by the computer. On computer-observer disagreements, a third experienced observer decided the final code. ST-segment classification was done on all 472 ECGs of this study. The final codes, decided by the third observer, are considered to be the correct ones.

Patterns that were judged as belonging to one of Punsar's types and hence got a "final code" were found in 59 of 472 subjects. There were eight falsely negative findings and eight falsely positive computer codes, as illustrated in Table 2.I.

Of the eight falsely positive ECGs, one had pre-excitation and one right bundle branch block (Fig. 2.9), two conditions when ST-segment coding is not done according to the Minnesota and the Punsar codes. The disagreements between the computer and the final code were borderline in the remaining six falsely positive as well as in the eight falsely negative ECGs (Fig. 2.9).

		FINAL CODE		Sum
		Present	Absent	
Computer code	Present	51	8	59
	Absent	8	405	413
Sum		59	413	472

Table 2.I. Number of ECGs with and without a final Punsar code, and the ability of the computer to identify them.

Obviously, in the codes describing minor ST-junction depressions of the order of 0.5 mm, errors in visual judgement or computer measurements are liable to occur. Even very small errors in P-R-segment or J-point amplitude will cause differences in e.g. the I₅, S₄ and R₃ codes (Fig. 2.8).

From these results the sensitivity and the specificity (Rautaharju et al 1976) of the computer to find and to exclude a Punsar code in leads V₄-V₆ can be calculated. The sensitivity was $51/59 = 86\%$, which means that the computer correctly identifies 86 % of the codable ECGs. The specificity was $405/413 = 98\%$, which means that the computer correctly identifies 98 % of the non-codable ECGs. The predictive value of an ECG with a computer code was $51/59 = 86\%$, while the predicted value of an ECG without a computer code was $405/413 = 98\%$. Furthermore, the error ratio (ER), as described by Rautaharju et al (1976), was $(8 + 8)/51 = 0.3$, which means that there was one subject with a false or missed code to each three subjects with a correctly identified code. We combined the use of the computer program with visual verification in order to reduce coding errors as recommended by Rautaharju et al (1980). From Fig. 2.9 it appears that the computer did in fact produce codes with considerable accuracy. Devia-

		COMPUTER															
FINAL CODE	Code	I					S				R			NC			
	Code	1	2	3	4	5	1	2	3	4	1	2	3	NC			
I	1																
	2																
	3																
	4				1	4	1			①							
	5					6				2					1		
S	1																
	2																
	3								11	1							
	4					2			2	14					4		
R	1																
	2								①								
	3								2	①			2	3			
NC							①		①	3			3		405		

Fig. 2.9. Number of agreements and disagreements in coding according to Punsar (1968) by computer and final code in 472 subjects. Only the lead with the most serious code in each subject was considered. The figure encircled by a solid line is a severe disagreement and figures encircled with a dotted line are moderate disagreements (according to Werner 1976). The other disagreements are borderline. The subject with the severe disagreement had a right bundle branch block and the subject, in which the computer coded S3 in a not codable ECG, had pre-excitation. NC = not codable (normal ST-segments).

tions between the computer code and the final code were in general small, in regard to the great difficulties illustrated by Fig. 2.8. Rautaharju's recommendation to verify computer codes is nevertheless warranted, as illustrated by our program's inability to recognize ventricular conduction defects.

2.6 ORTHOGONAL ECG

An orthogonal ECG (VCG) with the Frank lead system (Frank 1956) was taken on all subjects by means of the computer-assisted system described by Jonson et al (1976). The VCGs were taken in supine position and with the leads in the fourth parasternal intercostal space level, as recommended by Langner et al (1958). All VCGs have been scrutinized for signs of disease according to well-known criteria (Benchimol 1979, Arvill et al 1972, Starr et al 1974, Cowdery et al 1980), without consideration to the scalar ECG. The VCGs have not been used for selection of subjects. On the assumption that the VCG is a somewhat more sensitive method than ECG for some kinds of heart disease, the VCGs were used in order to study if such detectable disease was prevalent in the material to an extent that would influence the normal limits (chapter 8). The VCGs was also used to shed some additional light upon uncommon ECG-patterns found in this material. A detailed analysis of the VCGs will appear in a separate study.

3 THE ELECTRICAL POSITION OF THE HEART AND THE LEAD SYSTEM

3.1 INTRODUCTION

Variations in the anatomical position of the heart will obviously affect the shape and size of the ECG-deflections. Therefore, attempts have been made to determine the position of the heart, in order to obtain a better evaluation of the ECG in a particular individual. Unfortunately, the anatomical position of the heart is not easy to define and to determine. One is therefore restricted to analyse the so called electrical position of the heart, which was described by Einthoven, Fahr and de Waart in 1913, as the direction of the resultant potential difference in the heart. Later, the term electrical axis was used. The electrical axis is the mean value of the frontal projection of all instantaneous vectors of the QRS-complex. It corresponds to the direction of the major electrical forces in the frontal plane. The electrical axis was calculated from the three standard leads I, II and III, which were the only leads originally available. With the introduction of unipolar leads (Wilson et al 1934) and augmented unipolar leads (Goldberger 1942), the electrical position of the heart could be classified more easily. Thus, Wilson et al (1943) suggested five different positional groups in the frontal plane. Differences in position in the frontal plane are regarded mainly as representing rotation around the anteroposterior axis. The electrical position of the heart, however, is a spatial entity. Position in the frontal plane represents only one of its three projections. Goldberger (1953) also considered the rotation around the long axis of the body (superior-inferior) and the transverse axis (right-left). Unfortunately, his classification system resulted in nine different groups. Such a large number of positional groups is cumbersome to use in clinical work. Attempts to reduce the number of subgroups increased the subjects with more diverse directions in each group. Thereby the gain in subgrouping was lost.

The electrical heart position is obviously not synonymous with the anatomical position. The relationship between the two positions varies with i.e. body build and transmission properties of the tissues. Furthermore, the relationship between the direction of the major electrical forces and the anatomical position is difficult to analyse due to the complex pattern of excitation when the major forces are produced 30 to 50 msec after the start of depolarization. It is possible that the direction of electrical forces during the initial (septal) phase of depolarization is more closely related to the anatomic position of the heart, notably the position of the septum. Therefore a method to define the electrical heart position from the initial septal vector - corresponding to the normal Q-wave - will be introduced in this study.

3.2 THE LEAD SYSTEM

In order to discuss the electrical position of the heart, the relationship between the 12 standard leads must be understood. The scalar leads may be regarded as describing the electrical activity of the heart in two planes, perpendicular to each other. The extremity leads I, II, III, aVR, aVL and aVF define a frontal plane. To simplify the analysis, the chest leads V_1 and V_6 are also regarded as situated in a single horizontal plane.

The extremity leads are often analysed in comparison with a geometrical analogue in the form of Einthoven's equilateral triangle (Fig. 3.1), with the heart in the center (Einthoven et al 1913, Goldman 1964). The bipolar leads I, II and III then form the sides, and the augmented bipolar leads the heights. Since the size of the deflections in a particular lead depends upon the parallel projection of the electrical vectorial force upon that lead, the triangle model can be changed by parallel transfer of the sides so that they pass the center of the equilateral triangle. This changes the triangle into a hexa-axial model, in which the six leads are separated by 30° angles (Fig. 3.1).

Leads I, II, III, aVL and aVF have their "positive poles" downwards or leftwards, i.e. conforming with the main anatomic and electric axes of the heart. The deflections within the QRS are hence mainly positive in these leads. However, the "positive pole" of lead aVR points in the other direction (Fig. 3.1). It has been suggested by Cabrera and others (Wird-Sollereeder 1963, White 1971) that the polarity of lead aVR should be changed so that this lead conforms better with the other five. This lead, -aVR, was used in the present study. It has the positive end pointing in the same main direction as the other five, being located between lead I and lead II (Fig. 3.1). Lead -aVR gets a maximal deflection for forces parallel to 30° in the frontal plane.

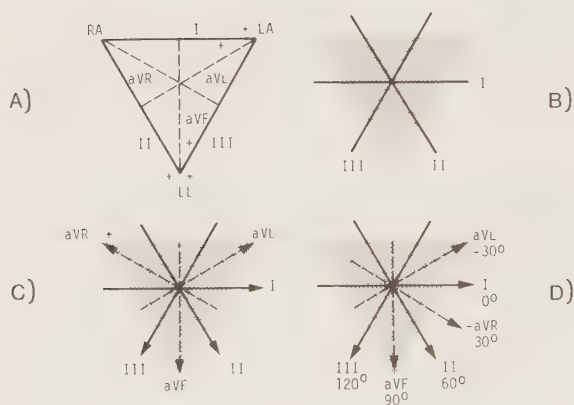


Fig. 3.1. A) The equilateral triangle of Einthoven, the sides of which represent the three bipolar leads I, II and III. The heights of the triangle represent the augmented unipolar leads aVL, aVR and aVF. + indicates the "positive pole" of the lead. The ECG-galvanometer deflects upwards when this pole is positive in relation to the other pole of the lead. B) Parallel transfer of the sides to the center of the triangle results in a tri-axial model frame for determination of the electrical position. C) The hexa-axial model is obtained when the augmented lead axes are added. D) The polarity of lead aVR has now been changed (see text).

RA = right arm, LA = left arm, LL = left leg

3.3 CONVENTIONAL DEFINITION OF THE ELECTRICAL POSITION

The mean frontal QRS-axis is the mean value of the frontal projections of all instantaneous vectors during ventricular depolarization. The direction of this vector is from the center of the heart towards the positive pole of that frontal plane lead with the largest net potential. To identify that lead may sometimes be difficult in practice, due to differences in size between the bipolar and the aV-lead deflections. It is easier to find the direction of the vector as being 90° from the lead with an equiphasic QRS-complex (Grant 1957), which has been done in this study.

Classification of the frontal position into subgroups can be made in various ways. Wilson (1943) originally used five groups according to the deflection pattern in the unipolar limb leads (VR, VL and VF). The five groups were: horizontal, semihorizontal, intermediate, semivertical and vertical. Simonson (1961) modified this, combining the horizontal and semihorizontal positions, and the vertical and semivertical positions. He then got three groups: horizontal (H), intermediate (I), and vertical (V). The basis for classification was still the deflection pattern in the unipolar or modified unipolar limb leads, especially aVL and aVF (Goldman 1964). However, classification may also be based on the direction of frontal QRS-axis. The definitions of the frontal positional groups in this study were: "horizontal" is to the left of intermediate, "intermediate" is $+30^\circ$ to $+59^\circ$, and "vertical" is to the right of intermediate, i.e. $+60^\circ$ or more.

The horizontal transitional zone is located in the precordial lead with equiphasic QRS-complex or between the two leads where the predominant polarity of the deflection changes. In the horizontal plane, the direction of the mean QRS-vector will usually be towards the positive pole of V₇ or V₈. As none of those leads are included in the standard ECG, the direction of the mean QRS-vector in the horizontal plane will have to be defined through the position of the transitional zone.

Simonson (1961) classified the positions in the horizontal plane in three groups: CM, CR and CL. CM (Chest Middle) corresponds to a transitional zone in V₃ or between V₃ and V₄. CR (Chest Right) indicates a position to the right of CM and CL (Chest Left) to the left of CM.

3.4 RESULTS

3.4.1 The mean frontal QRS-axis and age

The regression of the mean frontal QRS-axis (MFQRS-axis) with age was analysed in the accepted sample of subjects, with the following results:

MEN: MFQRS-axis = $88^\circ - 0.87 \times \text{AGE (years)}$
(SD = 26.2, $r = -0.35$, $p < 0.01$)

WOMEN: MFQRS-axis = $64^\circ - 0.24 \times \text{AGE (years)}$
(SD = 20.4, $r = -0.12$, $p = 0.09$)

In men, the MFQRS-axis shifts significantly to the left with age. From the regression equation above, the mean value for men at the age of 20 can be calculated to 71° and at the age of 60 years to 36° .

In women, the decrease of the MFQRS-axis with age was not statistically significant.

The more pronounced age variation in men may be explained i.a. by the presence of a factor which co-varies with age in men, but not in women. Such a factor is, for instance, the obesity index, which increases with age in men, but not in women (Table 1.VII). In other words, young women are as obese as older women in this study and therefore age variation of the MFQRS-axis in women is not reinforced by the variation with obesity. This could explain why age variation does not reach a statistically significant level in women.

The statistical relationship between age and obesity index and MFQRS-axis was estimated by multiple regression analysis (Appendix No. 1).

The distribution of mean frontal QRS-axis with age in men and women is illustrated in Fig. 3.2.

Left axis deviation $< 0^\circ$ (LAD) was found in five men, aged 31-47 years, and one woman, aged 57 years. In men, LAD was more frequent in the middle age group than among the older subjects. This is surprising, as one would expect LAD to be more common with advancing age (Ostrander 1967, Blackburn 1967, Simonson 1972). One explanation could be that LAD is a sign of heart disease, which is asymptomatic in younger age. In older age, however, when the symptoms appear, the subject would be eliminated from a study like the present one. Indeed, two men with LAD were excluded from this study because of suspected heart disease, and both of them were more than 50 years old.

Analysis of the VCGs of the five subjects with LAD revealed signs of infarction in three subjects (Nos. 3, 4 and 5 in Fig. 3.2). Well-known VCG-criteria of inferior infarction (Benchimol 1979, Arvill 1972) were found in subjects 4 and 5, and anteroseptal infarction in subject 3. Subject 1 had a QRS-duration of 0.11 s. He was admitted with subendocardial anterolateral infarction 6 months after he was examined in the present study. *In other words, four of the five men with LAD had probable sub-clinical heart disease and it is therefore reasonable to consider LAD, not only to be an aberrant ECG-feature, but also a sign of disease.* Subject 2 (MFQRS-axis of -45°) had an unusually leftward Q-axis with Q-waves only in leads aVL and I, and no Q-waves in precordial leads. This may be due to abnormal septal depolarisation, but could also reflect an extreme counter-clockwise rotation of the septum around the longitudinal axis of the heart and backward rotation of the apex around the transverse axis in a horizontal heart. This unusual heart position is a possible explanation of the LAD in this case. (fig 7.1. page 93)

The woman with LAD had a normal VCG. She was a heavy smoker with a blood pressure of 155/95 mm Hg and a weight of 86 kg (Fig. 3.2). Analysis of the VCGs of the subjects with a MFQRS-axis of 0° (eight men and two women) revealed unusual patterns in all cases, although signs of definite infarction were present only in two men (subjects 6 and 7, Fig. 3.2). The unusual patterns included anterior dominance (time as well as area), abnormal initial rotation with a figure of eight in the sagittal plane, and inferior and posterior defects. However, unusual heart position could not be excluded, especially in two cases with extreme leftward Q-axis, e.g. Q-waves present only in aVL and I, and absent in the precordial leads (compare with subject 2). ECGs and VCGs of subjects 3-7 are demonstrated in chapter 8, Figs. 8.1-8.5.

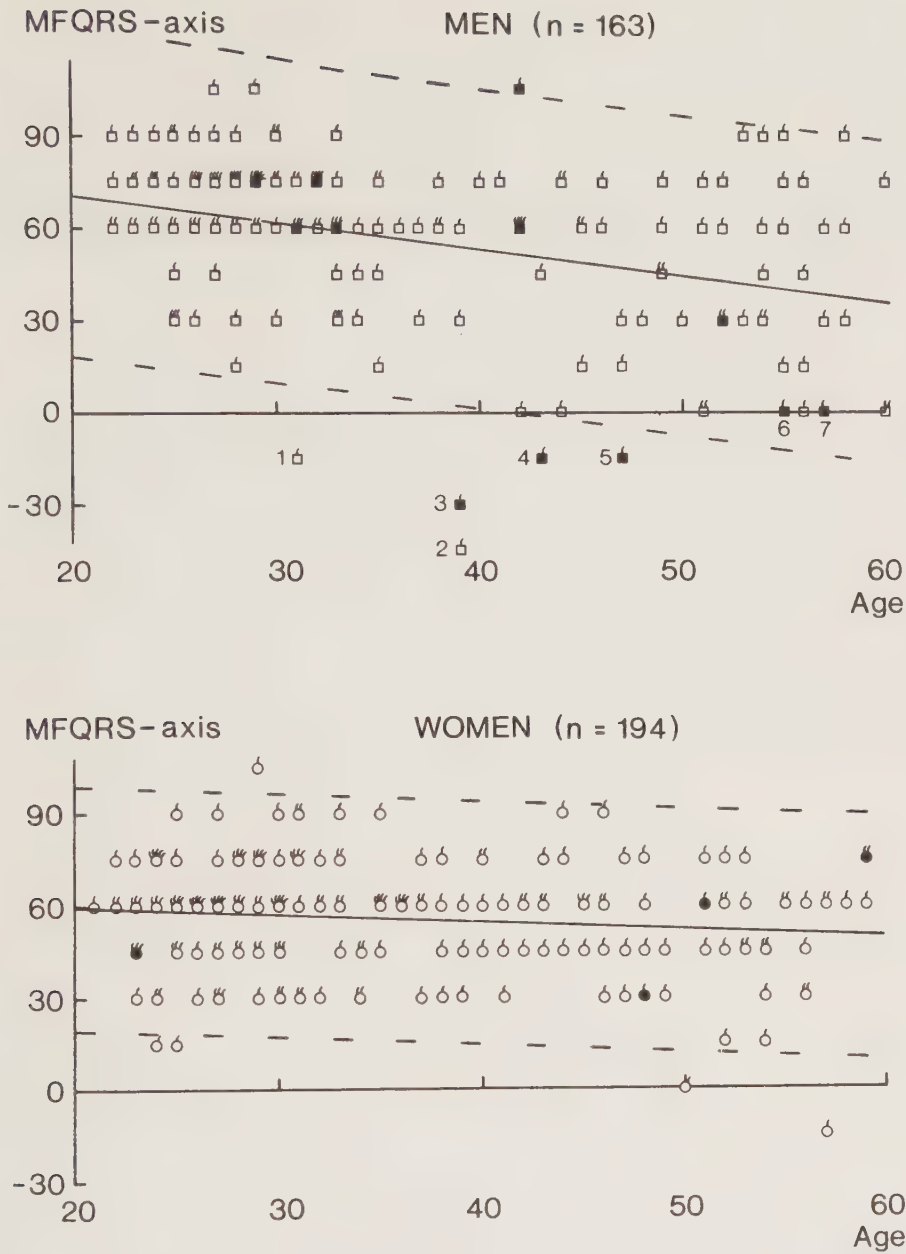


Fig. 3.2. The distribution of the mean frontal QRS-axis (MFQRS-axis) in men and women, aged 21-60 years. The limits of the 95 % confidence interval are noted. Subjects 1-7 among the men are commented upon in the text. Filled symbol indicates one subject with definitely or probably abnormal VCG (see chapter 8). Each tag on the symbol represent one individual.

VCG did not show the pattern of left anterior hemiblock (LAHB) in any of the subjects (men and women) with MFQRS-axis of zero degrees or less.

Mean values, standard deviation and normal limits as the 2.5 % and 97.5 % percentiles are reported in Table 3.I. The limits to the right were 90° in all ages, and in men as well as in women. The limit to the left was 15° in women of all ages. In men younger than 35 years the left limit was 30° and for men 35-60 years the limit was -15° . However, for the reasons given above, it is suggested that the left limit in older men should be changed to 0° .

Age		MEN	WOMEN
21-34 years	n	89	108
	M	65	56
	SD	21	22
	Lower	30°	15°
	Upper	90°	90°
35-60 years	n	74	86
	M	45	53
	SD	32	19
	Lower	-15° *	15°
	Upper	90°	90°

Table 3.I. The mean frontal QRS-axis in two age groups in men and women.

Mean values, SD and the 2.5 % and 97.5 % percentiles are given.

** The lower limit should probably be adjusted to 0° for reasons given on page 44 (see also Fig. 3.2).*

3.4.2 The mean frontal QRS-axis and blood pressure

The MFQRS-axis varied with systolic as well as diastolic blood pressure in men, but in women variation was only seen with diastolic blood pressure (Table 3.II). In women, the small influence of blood pressure on the MFQRS-axis may partly explain why the axis varies so little with age.

The statistical relationship between the MFQRS-axis and age and diastolic blood pressure was evaluated by multiple regression analysis and the results are given in Appendix No. 1.

3.4.3 The mean frontal QRS-axis and body build

Body build can be described in numerous ways (Simonson 1961). In this study, height, weight and chest circumference were measured. Body surface area and an obesity index were calculated. The relationship between the mean frontal QRS-axis (MFQRS-axis) and these anthropometric data were analysed with simple regression and the results are shown in Table 3.II. The results show that increases in weight, chest circumference and obesity index are all associated with a significant shift of the MFQRS-axis to the left in men as well as in women. On the other hand, no significant relationship was found between the MFQRS-axis and height or body surface area in man.

The results thus indicate that heavy subjects with a wide chest and a large obesity index are likely to have an MFQRS-axis to the left.

It has previously been shown that diastolic blood pressure influences the MFQRS-axis; the relative importance of blood pressure and body build on MFQRS-axis was therefore evaluated by multiple regression analysis, and the results are given in Appendix No. 1.

*Table 3.11. Regression of the mean frontal QRS-axis (MFQRS-axis) on systolic blood pressure (SBP) in mm Hg, diastolic blood pressure (DBP) in mm Hg, height [H] in cm, weight [W] in kg, chest circumference (CC) in cm, body surface area [BS] in m² and obesity index (OI) as W/H³. SD = standard deviation, r = correlation coefficient. Significance is reported as * = $p < 0.05$, ** = $p < 0.01$ and *** = $p < 0.001$.*

MEN

Equation	SD	r
MFQRS-axis = $114 - 0.45 \times \text{SBP}$	27.6	-0.18*
MFQRS-axis = $182 - 1.60 \times \text{DBP}$	25.3	-0.43***
MFQRS-axis = $-64 + 0.68 \times \text{H}$	27.7	0.15
MFQRS-axis = $111 - 0.75 \times \text{W}$	27.2	-0.24**
MFQRS-axis = $208 - 1.6 \times \text{CC}$	26.3	-0.33**
MFQRS-axis = $107 - 27.1 \times \text{BS}$	27.8	-0.12
MFQRS-axis = $138 - 6.3 \times \text{OI}$	26.1	-0.36**

WOMEN

Equation	SD	r
MFQRS-axis = $75 - 0.17 \times \text{SBP}$	20.2	-0.08
MFQRS-axis = $91 - 0.46 \times \text{DBP}$	20.0	-0.16*
MFQRS-axis = $32 + 0.14 \times \text{H}$	20.5	0.03
MFQRS-axis = $85 - 0.50 \times \text{W}$	20.1	-0.22**
MFQRS-axis = $104 - 0.56 \times \text{CC}$	20.2	-0.20*
MFQRS-axis = $102 - 27.9 \times \text{BS}$	20.3	-0.17*
MFQRS-axis = $94 - 2.9 \times \text{OI}$	19.9	-0.26**

3.4.4 The transitional zone in the chest leads

The influences of age, body build, blood pressure and MFQRS-axis on the position of the transitional zone have also been studied, but there were no significant differences between the three positional groups (CR, CM and CL, see page 43). However, there was a significant sex difference ($\chi^2 = 14.0$, $p < 0.001$, 2 d.f.), the CL-position being rare in women (found in only 6 out of 194, i.e. 3 %). On the other hand, the CR-position was more common in women than in men (Table 3.III).

	MEN	WOMEN
CR	49	82
CM	94	106
CL	20	6
Sum	163	194

Table 3.III. Number of men and women in the precordial positional groups CR, CM and CL.

No subjects (men or women) had the transitional zone in lead V_5 or leftwards. Eight subjects (4 men and 4 women) had the transitional zone to the right of lead V_2 . Analysis of the VCGs of these subjects revealed abnormal or probably abnormal QRS-loops in three of these subjects (see chapter 8).

Thus, normal limits of the transitional zone in the precordial leads for men and women are lead V_2 , and between leads V_4 and V_5 .

4 DEPOLARIZATION DEFLECTIONS

4.1 The Q-WAVE

The depolarization of the septum causes the initial part of the QRS-complex, i.e. the Q-wave in leads facing the left ventricle. Normally (Durrer et al 1970), septal depolarization starts on the left ventricular side of the septum. Excitation then proceeds towards the right chamber and the resultant force is directed from left to right. The Q-wave is described in terms of duration and amplitude.

Q-waves are not distributed randomly among the leads, but usually follow a strict distribution pattern. Different Q-wave patterns are likely to represent different positions of the septum. The use of the initial vector as a marker of heart position may offer some advantages in comparison with the conventional electrical axis. For example, electrical events in the beginning of the depolarization are not so complex, as the rest of the heart is electrically "silent" at this time. Therefore, the concept of "Q-axis" will be introduced and its possible role as indicator of heart position will be discussed.

4.1.1 Q-wave patterns

a) Men

The Q-wave pattern was analysed in the men of the accepted sample (n=163). The most common patterns in the chest leads were:

No Q-wave	in 8 ECGs (5 %)
Q-wave only in V_6	in 18 ECGs (11 %)
Q-waves in V_5 and V_6	in 68 ECGs (42 %)
Q-waves in V_4 , V_5 and V_6	in 54 ECGs (33 %)
Q-waves in V_3 , V_4 , V_5 and V_6	in 8 ECGs (5 %)
	156 ECGs (96 %)

Thus 96 % (156/163) of the men had one out of five Q-wave patterns in the precordial leads. The corresponding patterns in the extremity leads are reported in Table 4.I. This table shows that the Q-waves were not distributed at random but were present in successive adjacent leads. Furthermore, a large number of Q-waves in the extremity leads were associated with a large number of Q-waves in the precordial leads and vice versa. For example, in ECGs with five or six frontal Q-waves, there were usually three or four horizontal plane Q-waves. On the other hand, in ECGs without horizontal plane Q-waves, there were very rarely more than two frontal plane Q-waves. This may indicate that absence of Q-waves in the precordial leads is a normal finding in ECGs with few Q-waves in the extremity leads.

The remaining seven subjects had precordial Q-wave patterns not included in Table 4.I. These subjects will be discussed later.

Table 4.I. Q-wave combinations, found in 156 (96 %) of the men in the accepted sample. Thirty-three different combinations were represented. The number of subjects with each of the combinations is reported. Q-axis is explained in the text. QR, QI and QL indicate the direction of the Q-axis to be rightward, intermediate and leftward, respectively (Table 4.VII).

Q-wave combinations in the limb leads Precordial leads	III	aVF III	II aVF III	-aVR II aVF III	I -aVR II aVF III	aVL I -aVR II aVF III	aVL I -aVR II aVF III	aVL I -aVR II	aVL I -aVR	aVL I	aVL	Sum
No Q-wave	1		1							4	2	8
V ₆		1	5	6				1	3	2		18
V ₆ , V ₅	1		4	20	7	8	4	7	9	7	1	68
V ₆ , V ₅ , V ₄				11	13	7	8	6	6	3		54
V ₆ , V ₅ , V ₄ , V ₃				1	1	3	1	1		1		8
Sum	2	1	10	38	21	18	13	15	18	17	3	156
Positional group	QR 90° QI 0° QL											
Direction of the Q-axis	195°	165°	135°	105°	75°	45°	15°	-15°	-45°	-75°	-105°	

b) Women

The most common patterns in the horizontal plane among the women (n=194) were the same as in the men:

No Q-wave	in 4 ECGs (2.0 %)
Q-wave only in V ₆	in 30 ECGs (15 %)
Q-waves in V ₅ -V ₆	in 77 ECGs (40 %)
Q-waves in V ₄ -V ₆	in 68 ECGs (35 %)
Q-waves in V ₃ -V ₆	in 11 ECGs (5.5 %)

Five Q-wave patterns in 190 ECGs (98 %)

Thus, 98 % (190/194) of the women had one out of five precordial Q-wave patterns. The corresponding Q-wave patterns in the frontal plane leads are reported in Table 4.II.

The table indicates that also the female Q-waves are not distributed at random. The trend that a large number of Q-waves in the frontal plane is

associated with a large number in the horizontal plane held true also for women, although the trend was not as pronounced as in men. The absence of Q-waves in the horizontal plane was less common in women. The suggestion that absence of Q-waves in the precordial leads could be a normal phenomenon when associated with a low number of Q-waves in the frontal plane may also be applicable to women. Four women had precordial Q-wave patterns other than those in Table 4.II and they will be discussed next.

Table 4.II. Q-wave combinations, found in 190 (98 %) of the women in the accepted sample. Thirty-four different combinations were represented and the number of subjects with each of the combinations are reported. QR, QI and QL indicate the direction of the Q-axis to be rightward, intermediate and leftward, respectively (see Table 4.VII).

Q-wave patterns in the limb leads												
Precordial leads	III	aVF III	II aVF III	-aVR II aVF III	I aVR II aVF III	aVL I II aVF III	aVL I II aVF III	aVL I II aVF III	aVL I II aVF III	aVL I II aVF III	aVL I II aVF III	Sum
No Q-waves			1							2	1	4
V ₆ only	2		11	4		4		1	3	4	1	30
V ₆ , V ₅	1		8	29	7	6	4	7	7	7	1	77
V ₆ , V ₅ , V ₄			5	16	16	10	8	8	4		1	68
V ₆ , V ₅ , V ₄ , V ₃				2	2		3	2	1	1		11
Sum	3		25	51	25	20	15	18	15	14	4	190
Positional group	QR 90° QI 0° QL											
Direction of the Q-axis	195°	165°	135°	105°	75°	45°	15°	-15°	-45°	-75°	-105°	

c) Unusual Q-wave patterns

The Q-wave patterns in the eleven subjects not included in Tables 4.I and 4.II are reported in Table 4.III. It can be seen in this table that unusual Q-wave patterns are only seen in the precordial leads. The precordial Q-waves were all very small or sometimes appeared as an initially notched QS-complex when they were present in V₁. The precordial Q-wave patterns could be classified into three groups, namely those with a Q-wave in lead V₁ but not in V₆, those with a Q-wave in leads V₁ and V₆ and those with one or more Q-waves in any other precordial lead but not in V₁ and V₆. These three groups are indicated by horizontal lines in Table 4.III.

Analysis of the VCGs of the first group (subjects A-D) demonstrated an initial vector directed backwards to the left in 3 subjects (A-C), which VCGs were classified as abnormal (A and B) or probably abnormal (C). Subject D had an initial vector straight to the left (i.e. along the X-axis) and was classified as probably normal. The difference between the VCG of subject D and the VCG of subjects E-H, which were classified as normal, was one of degree only. Subjects E-H had slightly leftward but anteriorly directed initial vectors. Subjects I-L had normal VCGs with initial vectors directed to the right. They were of very low amplitude - a factor that may explain why detectable Q-waves were only present in some leads to the left but not in V₆.

The significance of Q-waves in V₁ is probably varying in subjects with and without Q-waves in V₆. Q-waves in V₆ indicate the presence of normal septal vectors. In these subjects the Q-waves in V₁ may represent local electrical forces close to the heart surface. Absence of Q in V₆ and presence of Q in V₁ may be concomitant signs of abnormal septal vectors as in septal fibrosis or infarction as was indicated by VCG (Benchimol 1979).

Table 4.III. Unusual Q-wave patterns in 7 men and 4 women, not included in Tables 4.I or 4.II.

			Q-wave patterns		VCG diagnosis
Ind	Sex	Age	Frontal	Horizontal	
A	M	39	aVL, I	V ₁ , V ₂ , V ₃ , V ₄ , V ₅	Anteroseptal infarction
B	M	42	III, aVF	V ₁ , V ₂	Anteroseptal infarction
C	W	59	III	V ₁	Septal fibrosis
D	M	55	III, aVF, II	V ₁	Probably normal
E	M	31	III, aVF, II	V ₁ , V ₅ -V ₆	Normal
F	W	46	III, aVF, II	V ₁ , V ₄ -V ₆	Normal
G	W	33	III, aVF, II	V ₁ , V ₄ -V ₆	Normal
H	M	39	aVL, I	V ₁ , V ₆	Normal
I	W	54	III, aVF, II	V ₄	Normal
K	M	35	aVL, I	V ₃ , V ₄	Normal
L	M	27	III	V ₂ , V ₃	Normal

4.1.2 Q-wave amplitude and duration

The Q-wave amplitude was found to decrease with age, but the decline was too small, however, to have any clinical importance. The results will be reported separately for men and women.

The Q-wave amplitude did not exceed 3 mm in any lead in either men or women, as demonstrated in table 4.IV (page 54). The Q-wave duration (Table 4.V) did not exceed 0.03 s, except in leads aVL (4 men) and III (2 women and 4 men), where the upper limits were found to be 0.04 s in men. Under certain circumstances such a long duration qualifies the ECG for Minnesota

code 1.3.3 or 1.3.4 (see footnote below).

In the two women and four of the eight men, the Q-wave was codable according to the Minnesota code. The prevalence of Q-wave items was 1.4 %. Code 1.3.3 was found in only one man while code 1.3.4 was found in two women and three men. No other Q-wave items were found in this study.

The VCGs of four subjects with a codable Q-wave in lead III were essentially normal although inferior vectors were small in three of them. One woman, on the other hand, had probable antero-apical infarction. The VCG of the man with a codable Q-wave in aVL had signs of lateral myocardial infarction. The results of the VCG analysis are compatible with the significance of Minnesota code 1.3.3. The relevance of code 1.3.4 appears to be more doubtful, at least if judged from the VCGs.

The Q/R-amplitude ratio has been analysed in all leads except V_1 and V_2 and the results are shown in Table 4.VI.

The QR-ratios were smallest in the precordial leads V_3 - V_5 and largest in leads aVL and III. In leads I, -aVR, II, aVF and V_6 , intermediate values were obtained. The Q/R-ratio exceeded 1/4 in leads aVL (54 subjects) and III (16 subjects), and most often in notched QRS-complexes of small total amplitude. In the other leads the Q/R-ratio exceeded 1/4 only in one man. He had a small notched complex in lead aVF and a mismatch of the frontal Q- and QRS-axes (see 4.1.3.b), suggesting loss of inferior vectors. His VCG, however, did not fulfill the criteria of inferior infarction. No important sex differences were found.

Considering the results above, *the normal Q-wave* can be described in the following terms.

1. It is present in adjacent leads covering 180° in any plane chosen.
2. A qrS or a QS may be present in lead V_1 if a normal Q-wave is present in lead V_6 .
3. Its amplitude should not exceed 0.3 mV.
4. Its duration should not exceed 0.03 s.
5. The Q/R-ratio should not exceed 1/4, except in leads aVL and III.

Code 1.3.3 is a Q-wave duration of at least 0.03 s but less than 0.04 s in aVL, provided the R-wave amplitude is 3 mm or more.

Code 1.3.4 is a Q-wave duration of at least 0.03 s, but less than 0.04 s in lead III, provided the Q-wave amplitude in lead aVF is at least 1 mm.

Table 4.IV. Q-wave amplitude (mm) in men and women. 1 mm = 0.1 mV. Upper limit is the 97.5 % percentile. The mean values refer to leads with a Q-wave.

SEX \ LEAD		aVL	I	-aVR	II	aVF	III	V ₃	V ₄	V ₅	V ₆
MEN n=163	n	86	105	123	116	103	95	11	64	132	150
	M	0.8	0.7	0.6	0.8	0.8	1.1	0.4	0.6	0.9	0.9
	Upper limit	2.6	1.8	1.5	2.1	2.1	2.9	1.5	2.4	2.9	2.6
WOMEN n=194	n	85	107	142	157	141	130	12	82	158	186
	M	0.6	0.6	0.5	0.7	0.7	0.9	0.3	0.5	0.6	0.7
	Upper limit	2.0	1.7	1.3	1.8	2.0	2.4	1.1	1.9	1.9	1.7

Table 4.V. Q-wave duration (s) in men and women. Upper limit is the 97.5 % percentile rounded off to the nearest 0.01 s above. The mean values refer to leads with a Q-wave.

SEX \ LEAD		aVL	I	-aVR	II	aVF	III	V ₃	V ₄	V ₅	V ₆
MEN n=163	n	87	105	124	116	103	95	12	67	132	150
	M	.018	.016	.016	.017	.017	.019	.009	.012	.016	.017
	Upper limit	0.04	0.03	0.03	0.03	0.03	0.04	0.02	0.03	0.03	0.03
WOMEN n=194	n	85	107	144	161	142	132	13	83	159	188
	M	.015	.014	.014	.014	.016	.017	.008	.011	.014	.015
	Upper limit	0.03	0.02	0.02	0.03	0.03	0.03	0.02	0.03	0.02	0.02

Table 4.VI. Mean values and upper limit as the 97.5 % percentile of the Q/R-amplitude ratio in men and women in 10 standard leads.

SEX \ LEAD		aVL	I	-aVR	II	aVF	III	V ₃	V ₄	V ₅	V ₆
MEN	n	84	105	123	116	103	95	11	64	132	150
	Mean	.27	.10	.066	.070	.080	.12	.030	.028	.055	.077
	Upper limit	1.20	0.25	0.20	0.20	0.25	0.40	0.10	0.10	0.20	0.20
WOMEN	n	83	107	142	157	141	130	12	82	158	189
	Mean	.23	.083	.063	.062	.081	.15	.024	.032	.050	.066
	Upper limit	1.00	0.20	0.20	0.20	0.20	.60	0.20	0.20	0.20	0.20

4.1.3 The Q-axis

- a) The Q-axis and its possible role as an indicator of the heart position

In order to understand why a certain Q-wave pattern corresponds to a certain position of the heart, a brief repetition of the "vectorial approach" to ECG (Grant 1957, Constant 1981) is necessary.

For a theoretical analysis of the ECG, the human body can be regarded as a homogenous cylindrical volume conductor with a central dipole that represents the size and direction of the electrical vector force. Theoretic and experimental studies suggest that this assumption is reasonably valid (Grant 1950). In the electrical field of such a conductor, a plane of zero potentiality - the null plane - extends perpendicular to the vector, and from its center (Fig. 4.1). The line of zero potentiality, where the plane intersects the surface of the cylinder, is called the null contour. This contour divides the surface into areas of positive and negative potentials. The location of the null contour is determined by the position and direction, but not by the magnitude, of the central vector. If the null contour is known, one can calculate the direction of the vector, which is perpendicular to the null plane and points towards the area of positive potential.

The two concepts, the central vector and the null contour have counterparts in human electrocardiography. The central vector is analogous to the spatial QRS-vector. In 1950, it was shown by Grant that the null contour of the mean spatial QRS-vector coincided with the location of equiphasic QRS-complexes in multiple unipolar leads from all surfaces of the chest. Grant also found that all deflections on the "positive" side of the null contour were net positive, while those on the other side were net negative. The same rules also apply to the initial vector. Positive deflections appear on the "positive side" and negative (i.e. Q-waves) on the negative side. In Fig. 4.2 the initial(septal)vector is placed in a simplified model of the human torso. From this figure, it can be seen that different Q-wave patterns correspond to different directions of the initial vector. In order to facilitate classification of the Q-wave patterns, the concept "Q-axis" will be introduced. The Q-axis is the Q-wave pattern, expressed as a single figure. The Q-axis is defined as the axis of the septal vector, but with the opposite direction, i.e. pointing towards the Q-waves.

The direction of a frontal Q-axis can be calculated from the Q-wave "transitional zone" in the extremity leads. The "transitional zone" for the Q-wave is located between the two leads, where the Q-wave disappears. The direction of the Q-axis is 90° from this transitional zone (Fig. 4.3). A reasonably valid estimate can also be made from a mere inspection of the ECG. Many ECGs have an intermediate Q-axis (Table 4.VII) with Q-waves in 5 or 6 frontal leads. Leftward deviation of Q-axis is associated with disappearing Q-waves in lead III and adjacent leads. Rightward deviation is associated with disappearance of the Q-wave in aVL and adjacent leads.

Table 4.VII. Labels of the three positional groups according to the direction of the frontal Q-axis and characteristic features in the ECG.

Label of positional group	Mean frontal Q-axis	Number of Q-waves in extremity leads	Q-wave in aVL	Q-wave in III
Q-Left (QL)	$< 0^{\circ}$	1-4	+	-
Q-Intermediate (QI)	$90-0^{\circ}$	5-6	±	±
Q-Right (QR)	$> 90^{\circ}$	1-4	-	+

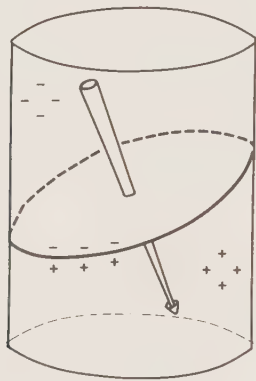


Fig. 4.1. Schematic drawing of a vector (thick arrow) and its null plane in a cylinder. The null plane is perpendicular to the vector, and indicated as the null contour (thick line) where it intersects the surface of the cylinder. The null plane is the plane of zero potential dividing the cylinder into a "positive" and a "negative" part.

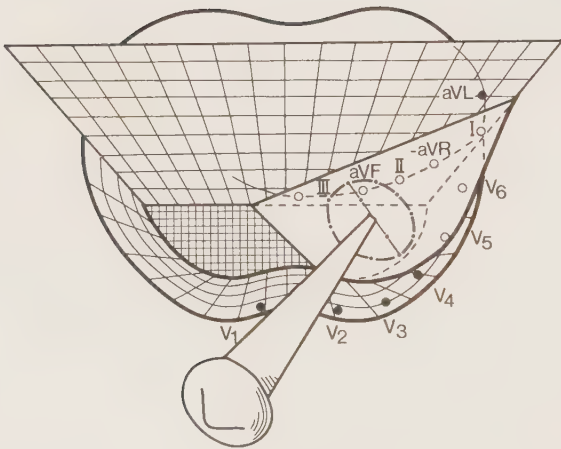


Fig. 4.2. The septal depolarization vector and its null plane in a simplified model of the human torso. Hypothetical position of septum in the null plane and the long axis of the heart are inserted (—•—). Symbols for the lead points of the frontal and horizontal planes are indicated, and those lead points situated inferiorly-posteriorly to the null plane (leads III-I and V₅-V₆) will show a Q-wave, while those on the "positive side" of the null plane (aVL, V₁-V₄) will not. If, however, the vector is moved to the right, posteriorly and inferiorly, Q-waves will appear in aVL and V₄, and the Q-wave in lead III will disappear.

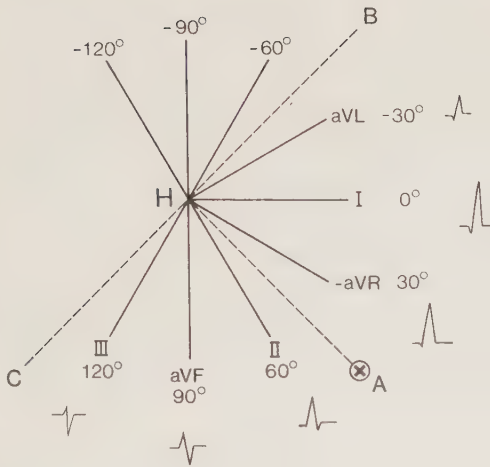


Fig. 4.3. Guide to calculation of the Q-axis. Point A represents the Q-wave transition, positioned between the two leads where the Q-wave disappears. In the figure, the Q-wave transition is at 45° . The direction of the Q-axis (B) is perpendicular to axis A-H towards the leads with a Q-wave. In the figure, the Q-axis has a direction of -45° . The direction of the Q-axis is opposite to the septal vector (C) by definition. H = electrical centrum of the heart.

b) The Q-axis in relation to the QRS-axis

The relationship between the Q-axis and the mean QRS-axis has been investigated in the frontal plane with regression analysis in men and women, and these were the results:

MEN

$$\text{Frontal Q-axis} = -63^\circ + 1.80 \times \text{MFQRS-axis} \quad (\text{SD} = 60.2, r = 0.64, p < 0.001)$$

WOMEN

$$\text{Frontal Q-axis} = -57^\circ + 2.40 \times \text{MFQRS-axis} \quad (\text{SD} = 65.0, r = 0.54, p < 0.001)$$

The regression analysis demonstrates a statistically significant relationship between the frontal Q-axis and QRS-axis in men as well as in women. The standard deviation for the Q-axis is much larger than for the MFQRS-axis. The reason for this may be that the Q-axis is measured with an accuracy of 30° compared to 15° for the MFQRS-axis. It is also possible that some Q-waves are missed because of very low amplitude, such as in subjects I-L in Table 4.III. Thirdly, it is reasonable to assume that the Q-axis is directed nearly perpendicular to the septum and the QRS-axis is directed more towards the apex of the heart, as illustrated in Fig. 4.4. If so, a rotation of the heart around its long axis would result in a wider range of possible Q-axes, compared to QRS-axes. The hypothetical directions of these axes in three hearts with different heart positions are illustrated in Fig. 4.4. It can be seen that a counterclockwise (CCW) rotation in the frontal plane was accompanied by a CCW-rotation around the long axis of the heart. The combined rotation was particularly pronounced in horizontal hearts and this has also been found in other studies (Meek 1925, Dougherty 1970). The pattern is the same in women.

It is interesting to note the relative position of the left and right ventricles in the vertical heart. As illustrated in Fig. 4.4, it ought to be possible that the right border of the radiologic cardiac silhouette is formed by the left ventricle in some vertical hearts with a Q-axis in QR-position. This would be in line with the findings of Goldberger (1949).

It is also interesting to note that the frontal directions of the Q-axis and QRS-axis are close to each other in vertical and intermediate positions, while they diverge in direction in the horizontal position. One could speculate over, whether a mismatch of the Q- and QRS-axes implies loss of vectors in either the septum (Q-axis) or the free wall of the left ventricle (QRS-axis). For instance, if the Q-axis is 90° , one would expect the QRS-axis to be about the same (Fig. 4.4). If, however, the QRS-axis is horizontal (say $+30^\circ$), it is quite possible that this may indicate loss of inferior vectors in the free left ventricular wall.

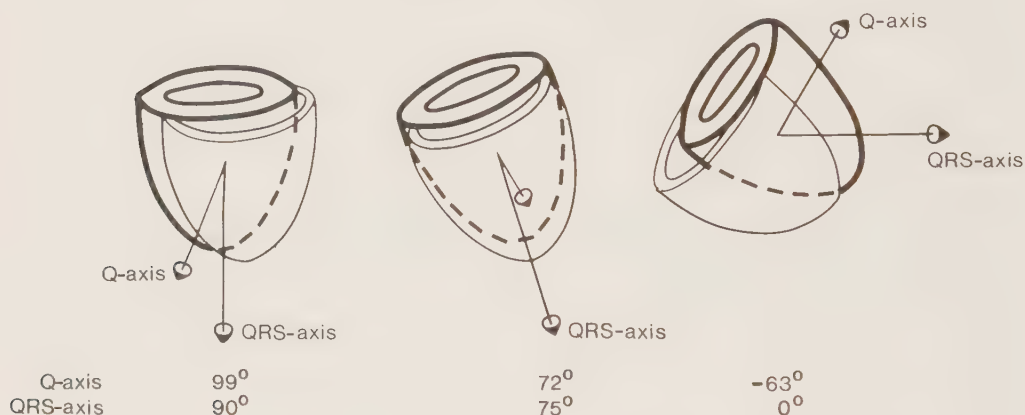


Fig. 4.4. Schematic drawing of the heart, seen from the front, showing the combined rotation of the heart in men. The apex of the heart is pushed backwards in order to better demonstrate the left and right ventricles. A CCW-rotation around the antero-posterior axis is associated with a CCW-rotation around the longitudinal axis of the heart (seen from apex). Thick line = left ventricle. The angle values are derived from the regression equation for men on previous page.

4.2 The R-WAVE

Measurements of the R-wave amplitude have been made in all 12 leads. Age variation is found for most leads in men. In women, however, age variation is less pronounced and significant only in three leads (II, aVF and III). Stratification according to age will therefore not be done in women. R-wave amplitude varies with heart position in the lead (see 4.2.2) in both men and women expressed as presence or absence of a Q-wave.

The MFQRS-axis is as expected an important determinant of R-wave amplitudes in the limb leads as is the position of the transitional zone in the precordial leads. Appendices Nos. 2 and 3 give further details about influences on the R-wave amplitudes in men and women, respectively.

4.2.1 The R-wave amplitude and age

a) Men

Variation with age was found in most leads, as illustrated in Table 4.VIII. There was a decline of amplitude with age in leads -aVR to III and V₁-V₆, but an increase of amplitude with age in leads aVL and I. The age variation was significant in leads aVL, -aVR to III, V₁-V₂ and V₆. It was not significant in leads I and V₃-V₅.

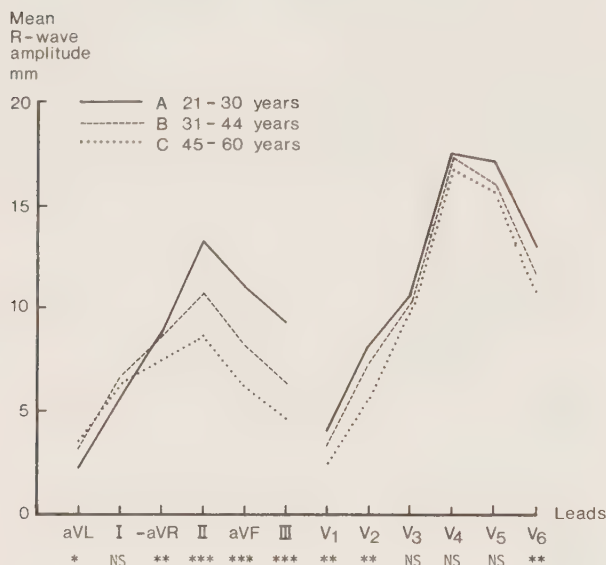
Table 4.VIII. R-wave amplitude in relation to age in men (n=163). Regression equations, standard deviation (SD), correlation coefficient (r) and significance of correlation [p-value] are reported. R-amplitude is given in mm, 1 mm = 0.1 mV. Age is given in years.

		SD	r	p-value
RH _{aVL}	= 1.2 + 0.042 x AGE	2.33	0.20	0.012*
RH _I	= 5.0 + 0.030 x AGE	2.84	0.12	0.14
RH _{-aVR}	= 10.3 - 0.054 x AGE	2.23	-0.26	< 0.001***
RH _{II}	= 17.1 - 0.16 x AGE	3.58	-0.46	< 0.001***
RH _{aVF}	= 15.0 - 0.17 x AGE	4.14	-0.42	< 0.001***
RH _{III}	= 12.8 - 0.16 x AGE	4.22	-0.39	< 0.001***
RH _{V₁}	= 5.1 - 0.048 x AGE	1.67	-0.31	< 0.001***
RH _{V₂}	= 9.9 - 0.075 x AGE	3.24	-0.25	0.0011**
RH _{V₃}	= 11.0 - 0.020 x AGE	4.65	-0.05	0.54
RH _{V₄}	= 18.3 - 0.031 x AGE	6.52	-0.05	0.49
RH _{V₅}	= 18.1 - 0.048 x AGE	5.15	-0.10	0.18
RH _{V₆}	= 14.8 - 0.078 x AGE	3.31	-0.26	< 0.001***

The fact that the R-wave amplitude increased with advancing age in aVL and I, and decreased in -aVR, II, aVF and III, reflects the shift to the left of the electrical axis with age in the frontal plane (see 3.4.1). However, the leftward shift of the axis was probably not the only cause of these variations, since the decrease of amplitude in leads II, aVF and III was far greater than the increase in leads aVL and I (Fig. 4.5). In this context, the overall decrease in R-wave amplitude with advancing age must be considered. The maximum spatial vector in VCG also decreased with age in this material and in the study by Pipberger et al (1967). This decline of R-wave amplitude was combined with the changes caused by the axis shift. Thus the decrease of amplitude in -aVR, II, aVF and III became enhanced, while the increase in aVL and I became less pronounced than it would be if the axis shift alone was responsible for the increase. The overall decrease in R-amplitude with age (Simonson 1961, Pipberger 1967, Benchimol 1979) may be due either to a decrease in myocardial mass or to reduced electrical transmission e.g. in the lungs and skin.

The R-wave was highest in the left precordial leads, especially V₄ and V₅. In young men it was also high in leads II and aVF. The smallest R-waves were found in leads aVL and V₁.

The height of the R-wave was subject to progressive changes from lead to lead, as illustrated in Fig. 4.5.



*Fig. 4.5. R-wave amplitude in men. Mean values (mm) of R-wave amplitude in men 21-60 years of age (n=163), subdivided into three age groups A, B and C. Values (from Appendix No. 2) are given for all twelve leads in the resting ECGs. 1 mm = 0.1 mV. Significance of the differences have been tested with F-test. *** = $p < 0.001$, ** = $p < 0.01$, * = $p < 0.05$. NS = not significant.*

b) Women

The R-wave amplitude did not vary significantly with age in women except in leads II, aVF and III (Table 4.IX). In these leads, there was a slight decrease of amplitude with age. As in men, the R-wave was highest in the precordial leads, especially V₄ and V₅. The smallest R-waves were found in aVL and V₁, as in men. The R-wave amplitude was also in women subject to progressive changes between the leads.

c) Sex differences in R-wave amplitudes

It is evident from this study, that the R-wave amplitudes were higher in men than in women. The magnitudes of these differences are reported in Table 4.X. The sex differences were most pronounced in younger ages (21-30 years) being present in leads II, aVF, III and V₁-V₆. With increasing age, however, these differences tended to diminish, and in age group C (45-60 years), statistically significant differences remained only in leads V₄-V₅.

Footnote: Peak-to-peak amplitudes in the 12 standard leads will be given in Appendix No. 6.

Table 4.IX. R-wave amplitude in relation to age in women (n=194). Regression equations, standard deviation (SD), correlation coefficient (r) and significance of correlation (p-value) are reported. R-wave amplitude is given in mm and age in years. 1 mm = 0.1 mV.

		SD	r	p-value
R_{aVL}	$= 1.7 + 0.017 \times \text{AGE}$	1.61	0.11	0.12
R_I	$= 5.2 + 0.019 \times \text{AGE}$	2.19	0.09	0.21
R_{-aVR}	$= 8.2 - 0.009 \times \text{AGE}$	1.94	-0.05	0.48
R_{II}	$= 11.9 - 0.040 \times \text{AGE}$	3.03	-0.14	0.05*
R_{aVF}	$= 9.9 - 0.051 \times \text{AGE}$	3.44	-0.15	0.03*
R_{III}	$= 8.0 - 0.058 \times \text{AGE}$	3.74	-0.16	0.02*
R_{V1}	$= 3.0 - 0.012 \times \text{AGE}$	1.34	-0.10	0.18
R_{V2}	$= 6.6 - 0.027 \times \text{AGE}$	2.53	-0.11	0.12
R_{V3}	$= 7.9 + 0.007 \times \text{AGE}$	3.75	0.02	0.78
R_{V4}	$= 11.2 + 0.050 \times \text{AGE}$	4.19	0.13	0.08
R_{V5}	$= 11.8 + 0.016 \times \text{AGE}$	3.73	0.05	0.53
R_{V6}	$= 11.1 - 0.023 \times \text{AGE}$	2.83	-0.09	0.23

Table 4.X. Magnitude of the differences in R-wave amplitude between men and women. Values given in the table are differences (mm) in mean R-wave amplitude between men and women ($R\delta$ - $R\varphi$), subdivided into three age groups (A, B and C). Symbols indicate significance according to Student's test. * = $p < 0.05$, ** = $p < 0.01$ and *** = $p < 0.001$.

	δ/φ	aVL	I	-aVR	II	aVF	III	V_1	V_2	V_3	V_4	V_5	V_6
A 21-30 years	n 66/ 83				***	***	***	***	***	**	***	***	***
		-.1	-.3	.7	2.1	2.4	2.6	1.3	2.0	2.2	4.6	4.6	2.3
B 31-44 years	n 51/ 63		*	*				**	***	**	***	***	**
		.7	1.2	.9	.5	.1	.4	.8	1.9	2.7	4.8	3.9	1.6
C 45-60 years	n 46/ 48										**	***	
		.7	.1	-.5	-1.2	-1.1	-.3	.1	.4	1.3	2.8	3.0	.9

4.2.2 The R-wave amplitude and heart position, as judged from the Q-wave

In the individual lead, the information of the electrical heart position is restricted to two alternatives. The lead point may either face the left ventricular side or the right ventricular side of the septum. The presence of a Q-wave in the lead indicates that the lead point is facing the left ventricular side and the absence of a Q-wave indicates that the lead point is facing the right ventricular side of the septum.

The size of a deflection in a particular lead is influenced by the position of the heart and by age. To demonstrate the influence of the heart position, the variance due to age must be reduced to a minimum, and this can be done by using the age corrected mean value of the R-wave amplitude, as calculated with simple regression analysis (see Table 4.VII). The age-corrected mean value of R-wave amplitude (RH_{AC}) is the expected value of the R-wave amplitude for a particular age. The observed R-wave amplitude (RH) may deviate from the RH_{AC} , due to factors other than age. This remaining variance is partly due to differences in heart position, which is reflected in the presence or absence of a Q-wave in the lead. The difference between the observed RH and the expected value of RH with respect to age (RH_{AC}) is therefore a measurement of the influence from other factors than age. The difference $RH - RH_{AC}$ has been calculated for each subject of the accepted sample in all standard leads except $V_1 - V_3$.

The mean values of the differences between $RH - RH_{AC}$ in these nine standard leads are plotted in Fig. 4.6 for men and women, differing with respect to the presence or absence of a Q-wave in the particular lead. The mean values of the Q-group indicate that the observed R-wave amplitudes were usually larger than the expected RH_{AC} . On the other hand, the mean values of the non-Q-group indicate that the observed R-wave amplitudes were usually smaller than RH_{AC} . The differences were significant in all the leads except in V_6 , men and women alike. Failure to demonstrate significance in this lead might be due to the low number of ECGs without a Q-wave in V_6 (13 men and 6 women). In the frontal plane, lead -aVR appeared less influenced by the heart position than the other frontal leads. The reason for this might be the fact that the spatial orientation of this lead axis parallels the most common direction of the MFQRS-axis. The projections of the major electrical forces will therefore not vary very much in this lead.

R-wave amplitudes in leads aVL-III and $V_4 - V_5$ with the presence of a Q-wave were nevertheless significantly larger than R-wave amplitudes in the same leads without a Q-wave. This stresses the importance to take not only sex and age, but also heart position, into consideration when delineating the normal limits of the deflection amplitudes (at least, then, the R-wave amplitudes).

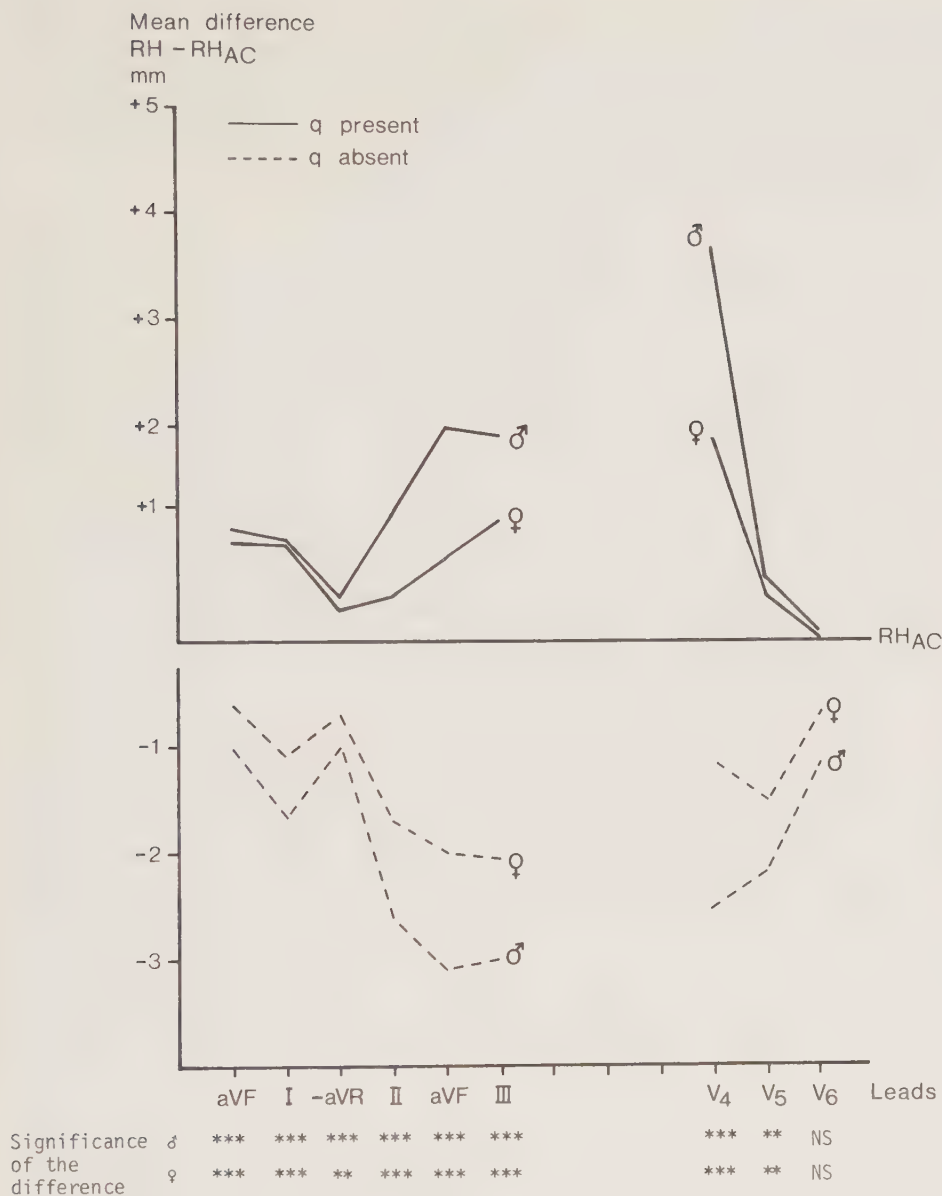


Fig. 4.6. The effects of the presence or absence of a Q-wave on the R-wave amplitude in nine standard leads. Mean values of the difference RH - RH_{AC} are plotted for the men (n=163) and women (n=194) of the accepted sample. Student's test was performed and significance of the differences are reported. * = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$, NS = not significant. RH = observed R-wave amplitude, RH_{AC} = expected R-wave amplitude as obtained from the regression R-wave amplitude/age (Tables 4.VIII and 4.IX).

Table 4.XI. R-wave amplitudes (mm) in men, with respect to age and heart position. 1 mm = 0.1 mV. Mean values, standard deviation (SD) and normal limits are reported in two age groups (Y and O), each of which have been divided into two positional groups, indicated by the presence or absence of a Q-wave in the lead. Significance of the differences (Students' test) between the positional groups are indicated. * = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$. UL = upper limit, LL = lower limit.

Age	Q-wave present		aVL	I	-aVR	II	aVF	III	V ₁	V ₂	V ₃	V ₄	V ₅	V ₆
Y 21-34 years n=89	Yes	n	37	54	71	68	63	58				37	71	89
		M	3.5	6.8	9.2	13.6	12.2	10.8				22.0	17.7	12.7
		SD	2.8	2.9	2.3	3.3	3.4	3.9				6.8	5.2	3.4
		UL	10.2	14.2	13.6	21.3	19.1	20.0				36.1	30.7	20.4
		LL	0.6	2.5	4.1	7.1	4.4	3.2				12.4	8.0	6.1
		Diff.	***	***	**	***	***	***				***	**	
	No	n	50	35	18	21	26	31	89	89	89	52	18	
		M	1.6	4.3	7.4	9.5	6.1	4.5	3.8	7.7	10.5	14.4	13.4	
		SD	1.2	1.9	1.5	2.4	2.5	2.5	1.8	3.2	5.0	6.0	4.1	
		UL	5.5	7.8	10.0	13.7	10.5	10.0	9.4	14.4	23.1	30.8	20.7	
		LL	0.4	1.6	4.9	5.2	1.3	1.0	1.2	2.1	3.5	5.0	6.1	
O 35-60 years n=74	Yes	n	47	51	52	48	40	37				27	61	74
		M	4.2	7.2	8.1	10.4	9.0	7.4				19.5	16.2	11.0
		SD	2.5	2.9	2.1	3.8	3.8	3.6				4.6	4.9	3.2
		UL	10.2	14.7	12.3	16.9	15.7	13.8				29.5	28.8	19.8
		LL	0.7	1.9	3.6	3.2	1.5	2.5				12.3	7.6	5.3
		Diff.	***	***	**	***	***	***				***	*	
	No	n	27	23	22	26	34	37	72	74	74	47	13	
		M	1.4	4.6	6.5	6.8	3.8	2.5	2.9	6.3	10.0	15.2	13.1	
		SD	0.72	1.5	2.0	2.1	2.2	1.6	1.5	3.4	4.2	5.2	4.8	
		UL	3.2	7.0	12.1	10.2	8.3	6.5	6.7	14.8	19.9	26.9	23.0	
		LL	0.2	1.5	4.1	3.4	0.8	0.9	0.9	1.1	2.6	6.6	7.7	

4.2.3 Normal limits of R-wave amplitude

a) Men

When delineating normal limits for men, one has to consider age as well as heart position (Table 4.XI). In leads V₁-V₃ and V₆, no grouping according to the presence or absence of a Q-wave has been done, because of the limited number of observations (too few subjects with Q in V₁-V₃, and too few without Q in V₆).

b) Women

The slight age variation in leads II, aVF and III observed with regression analysis in women did not result in differences when delineating normal limits as the 2.5 % or 97.5 % percentiles. However, heart position, as indicated by the presence or absence of Q-wave in the leads, significantly influenced the R-wave amplitude in the same leads, as in men Stratification of the R-wave amplitude values in women will therefore be done according to position, but not to age. The results is reported in Table 4.XII.

Table 4.XII. R-wave amplitude (mm) in women with respect to heart position, indicated by the presence or absence of a Q-wave in the lead. Mean values, standard deviation (SD) and normal limits are reported. Significance of the differences (Students' test) between the positional groups are indicated. * = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$. UL = upper limit, LL = lower limit.

Q-wave present		aVL	I	-aVR	II	aVF	III	V ₁	V ₂	V ₃	V ₄	V ₅	V ₆
Yes	n	83	107	142	157	141	130			11	82	158	194
	Mean	3.2	6.7	8.1	10.8	8.9	7.2			13.0	14.9	12.8	10.3
	SD	1.9	2.1	1.9	2.9	3.4	3.7			6.0	4.0	3.7	2.8
	UL	7.3	9.8	12.5	17.7	16.6	15.4			23.1	24.5	22.1	16.9
	LL	0.5	2.3	4.7	5.6	3.4	1.3			5.7	8.0	6.1	5.4
Students' t-test		***	***	**	***	***	***			***	***	**	
No	n	109	87	52	37	53	64	194	194	183	112	36	
	Mean	1.7	4.7	7.2	8.8	6.0	3.3	2.6	5.6	7.8	11.5	10.7	
	SD	0.94	1.7	1.8	3.2	2.8	2.1	1.3	2.5	3.3	3.8	3.4	
	UL	4.0	8.7	10.7	15.6	12.5	8.9	5.5	12.2	15.5	19.8	16.7	
	LL	0.3	1.3	3.4	3.9	1.0	0.8	0.6	1.6	2.8	4.1	5.6	

4.3 THE S-WAVE

Influence of age on the S-wave amplitude was demonstrated only in leads V₁ and V₂ in men. Stratification according to age will therefore not be done in the other leads. Stratification according to heart position will on the other hand be done in men as well as women. Heart position (presence or absence of a Q-wave in the lead) is a main determinant of the magnitude of the S-wave amplitude.

The S-wave in V₁ and V₂ and its relation to age will be analysed here, as it is of interest in connection with the diagnosis of left ventricular hypertrophy. Normal limits in relation to heart position will be reported in Appendix No. 4.

4.3.1 The S-wave amplitude in V₁ and V₂ and age

Regression with age has been analysed in leads V₁ and V₂. The regression equations, standard deviation (SD), correlation coefficient (r) and significance (p-value) were as follows:

MEN	SD	r	p-value
SV ₁ = 14.5 - 0.10 x AGE	4.18	-0.27	< 0.001***
SV ₂ = 26.4 - 0.25 x AGE	6.28	-0.40	< 0.001***
WOMEN			
SV ₁ = 9.8 + 0.01 x AGE	3.69	0.02	0.80
SV ₂ = 16.4 - 0.06 x AGE	5.45	-0.11	0.12

Men and women were subdivided into two age groups, namely young (Y), 21-34 years, and old (O), 35-60 years, and mean values, standard deviation and normal limits are given in Table 4.XIII.

The difference between the two age groups was particularly pronounced in lead V_2 , where the differences between the upper limits was 9.3 mm in men and 4 mm in women. Age stratification in leads V_1 and V_2 is important in view of the many criteria for hypertrophy suggested on the basis of S-wave amplitudes in these two leads. Ignoring the age-variation will result in low validity of the criteria, at least in men.

*Table 4.XIII. Influence of age on S-wave amplitude (mm) in leads V_1 - V_2 in men and women. Mean values, standard deviation (SD) and normal limits as the 2.5 % and 97.5 % percentiles are given in two age groups (Y and O). Significance of differences expressed with Students' t-value. *** = $p < 0.001$, NS = not significant.*

		MEN		WOMEN	
AGE	LEAD	V_1	V_2	V_1	V_2
Y 21-34 years	n	88	89	108	108
	M	11.8	19.8	9.9	14.7
	SD	4.5	7.3	3.8	5.6
	Upper	21.3	35.9	19.4	29.2
	Lower	4.2	4.3	3.4	5.7
O 35-60 years	n	72	74	85	86
	M	9.4	14.5	10.3	13.9
	SD	3.8	4.9	3.5	5.3
	Upper	18.8	26.6	17.8	25.2
	Lower	3.0	3.8	4.8	2.6
Students' t test		*** 3.6	*** 5.3	NS	NS

4.4 NOTCHING OF THE QRS-COMPLEX

Notching of the QRS-complex is a common finding in ECGs. It has, however, prognostic significance under certain circumstances (Weinberg et al 1950, Evans 1952). Weinberg et al (1950) stated that notching, in other leads than lead III, is almost invariably a sign of myocardial damage. They stated that notching is more likely to be significant if it occurs nearer the apex than the base of the complex, is present in multiple leads and is prominent. On the other hand, there is agreement that notching can normally occur near the so called transitional zone (Wilson et al 1943, Rosenman et al 1950). Evans (1952) found that notching in CR_4 - CR_7 was very rare, but when it occurred in clinically healthy subjects, the complex was of R_s -type. On the other hand, among patients with chest pain, he found several examples of notching in leads showing a qR -pattern.

Evans (1952) suggested the following definitions of notching: Notching may be initial, terminal or located in the mid-portion of the QRS-complex. It can be slight, moderate or prominent. The slight notching is defined as a slurring of one of the limbs of the deflection, i.e. an obvious local thickening. A moderate notching is characterized by a minor but definite brief spike somewhere on the QRS-complex (rR or Rr-type). A prominently notched deflection is splintered and has at least two apices (RR-type). It was believed by Evans (1952) that all three changes reflect the same mechanisms, namely an interruption in the progress of the electrical impulse through the myocardium, and that the distinction between them was only one of degree.

Notching in the present material

If the definition of notching is wide enough to include all types of notching occurring at all times of the QRS-complex, the prevalence in the present study was found to be very high. Two thirds of the QRS-complexes in leads aVL and III were notched and half of the complexes had a notch in lead aVF. One third had a notch in lead II, one fourth of the complexes were notched in leads I, V₁-V₃, and one fifth were notched in leads -aVR, V₄-V₆. Therefore, notching in this broad sense is too common to have any clinical usefulness. Detailed analysis of notching in the present study was therefore restricted to moderate or prominent notching of the R-wave in qR-complexes. By this method, notching will be studied only in true left ventricular complexes, well away from transitional zones in the frontal as well as the horizontal plane. Terminal notching, i.e. at the base of the downstroke of the R-wave, has not been considered, as this type of notching is likely to originate from a normal depolarisation of the outflow tract at the ventricular base.

Moderate or prominent notching of qR-complexes other than terminal was found in 15 % of the subjects (24 men and 28 women). The distribution of notched qR-complexes among the various leads is reported in Table 4.XIV. From this table it can be seen that notched qR-complexes are rare in leads I, -aVR, II and V₄-V₆. The ECGs with a notch in lead I were also notched in lead aVL, and the ECGs with a notch in -aVR and lead II were also notched in leads aVF and III. Furthermore, the ECGs with notching in V₄-V₆ also had notched complexes elsewhere in the frontal plane leads. There were no significant differences between men and women. The overall prevalence of notched qR-complexes is shown on the bottom line in Table 4.XIV.

Weinberg et al (1950) pointed out that notching was more likely to be significant if it occurred in multiple leads. Therefore, the number of subjects with notched qR-leads in at least two adjacent leads has been analysed. There were 15 men and 8 women (6.4 % of the subjects) with this feature. Furthermore, it is possible that notching in the beginning of the depolarisation is more significant than later notching, when individual variation of the excitation pattern is greater (Durrer et al 1970). Therefore, the prevalence of early notching among these 23 subjects has been analysed. Ten men and six women (4.5 % of the subjects in the accepted sample) were found to have early notching in at least one of the two notched and adjacent qR-complexes. A qR-complex with early notching had a qrR, qrsR or qRR-pattern, while later notching had a qRR-pattern. A notched Q-wave was given significance if the R-wave was also notched, and therefore a pattern of qqRr-type was noted as early notching, in spite of the fact that the R-wave was notched on the downstroke.

The VCGs of the 23 subjects with notched qR-complexes in two adjacent leads have been analysed. There were four abnormal or probably abnormal VCGs (17%) and three of them had early notching in at least one of the complexes. Such a high percentage of VCG abnormality is somewhat surprising, as one would not expect notching per se to result in a QRS-loop, which fulfills any of the present criteria for myocardial damage. It was also interesting to note that 10 of the 15 men (67 %) were heavy smokers. That is significantly more ($\chi^2 = 11.1$, $p < 0.001$, d.f. = 1) than in the remaining accepted male sample, in which 34/148 (23 %) were heavy smokers.

In a study with high fidelity ECG of patients with normal conventional ECG it was found that early notching, especially if present in several leads, predicted coronary artery disease (Sapoznikov et al 1977). It was also found that precordial leads were more sensitive in predicting the presence or absence of coronary artery disease than the limb leads.

Table 4.XIV. Prevalence of notched qR-complexes in men and women. Notching of the qR-complex was considered only when at least two obvious spikes were present on the R-wave, i.e. rR, RR or Rr-type. Terminal notching, that is late notching at the base of the R-wave downstroke, was not considered.

Lead Sex	aVL	I, -aVR	II	aVF, III	V ₁ -V ₃	V ₄ -V ₆
Men n=163	6 (3.7 %)	1 (0.6 %)	4 (2.5 %)	17 (10.4 %)	0	1 (0.6 %)
Women n=194	15 (7.7 %)	4 (2.1 %)	0	13 (6.7 %)	0	1 (0.6 %)
♂ + ♀ n=357	21 (5.9 %)	5 (2.2 %)	4 (1.1 %)	30 (8.4 %)	0	2 (0.6 %)

5 HIGH AMPLITUDE DEFLECTIONS AND LEFT VENTRICULAR HYPERTROPHY (LVH)

5.1 PROPOSAL OF NEW CRITERIA FOR LVH

In order to determine the limits between normal amplitudes and hypertrophy amplitudes, one has to examine the ECGs from subjects with normal hearts and compare them with those from patients with cardiac hypertrophy. This cannot of course be done in a study like this one, where all subjects are healthy. However, arbitrary limits can be set and the prevalence of ECGs exceeding those limits can be analysed. If that prevalence is sufficiently low in a healthy population, these criteria may be useful in the diagnosis of left ventricular hypertrophy.

A system designed to classify ECGs according to possible pathological significance should obviously not "pick up" too many healthy young men just because they have relatively high R-waves. On the other hand, the limits should not be set too high in relation to the lower amplitudes that are typical for women. In other words, there should be a good balance between specificity and sensitivity at all ages and in both sexes. Such a balance is impossible to achieve in systems where age and sex differences are ignored. However, it must also be borne in mind that a classification system must be easy to use.

The Minnesota code for left ventricular hypertrophy (items 3.1 and 3.3) is easy to use. Unfortunately, clinical experience suggests that these amplitude criteria provide too many falsely positive results.

Large amplitude R-waves according to the Minnesota codes 3.1 and 3.3 were found in 30 subjects (8.4 %). As shown in Table 5.I, the prevalence was higher in men than in women (14.1 % against 3.6 %; $\chi^2 = 11.4$, $p < 0.001$). The high prevalence in men was due to frequent high R-waves in lead V_5 in young men (age < 35 years). High frequency of hypertrophy criteria among the men was also seen with other classification systems (McPhie 1958, Mazzoleni et al 1964, Liu et al 1968, Romhilt and Estes 1968), as shown in Table 5.II.

Not only specificity, but also sensitivity, are known to be low for most of the current scalar ECG-criteria.

To overcome this deficiency, an attempt has been made in the present study to modify the amplitude criteria for left ventricular hypertrophy (Table 5.III). Consideration has been given, not only to variation with age and sex, but also to the electrical position of the heart.

Variation with age is statistically significant only in men and criteria limits have therefore been proposed for two age groups, namely 21-34 years and 35-60 years.

In women, on the other hand, limits have been set for all ages, i.e. 21-60 years, since the age variation was small.

Criteria for left ventricular hypertrophy were based upon the upper limits of R-wave amplitudes in left ventricular leads, that is leads with a Q-wave.

Table 5.I. Number of Minnesota code items 3.1 and 3.3, and number and prevalence of subjects with these codes, found in 357 healthy men and women. Young = 21-34 years, old = 35-60 years.

	MEN		WOMEN	
	Young n=89	Old n=74	Young n=108	Old n=86
Code 3.1				
RaVL > 12 mm	1			1
R _I > 20 mm				
R _{II} > 20 mm	2			
RaVF > 20 mm	1			
R _{III} > 20 mm	1			
R _{V5} > 26 mm	7	2	1	
R _{V6} > 26 mm				
Code 3.3				
R _I > 15 mm				
R _{V5} or R _{V6} + S _{V1} > 35 mm	6	5	4	1
Number of subjects with Code 3.1	10 (11.2 %)	2 (2.7 %)	1 (0.9 %)	1 (1.2 %)
Number of subjects with Code 3.3	6 (6.7 %)	5 (6.8 %)	4 (3.7 %)	1 (1.2 %)
Sum	23 (14.1 %)		7 (3.6 %)	

Table 5.II. Number and percentage of ECGs from healthy men and women qualifying for certain amplitude criteria of left ventricular hypertrophy. The criteria are from studies comtemporary to or later than the Minnesota code. I = Romhilt and Estes 1968, II = Mazzoleni et al 1964, III = Liu et al 1968, IV = McPhie 1958.

		♂ n=163	♀ n=194
I	S _{V2} > 30 mm	10	2
	R _{V5} > 30 mm	2	
	Sum	12 (7.4 %)	2 (1.0 %)
II	RaVL > 8 mm	7 (4.3 %)	1 (0.5 %)
III	R _{V6} ≥ 18 mm	10 (6.1 %)	2 (1.0 %)
IV	Max R + Max S > 45 mm (precordial leads)	19 (11.6 %)	1 (0.5 %)

Table 5.III. Proposed criteria for left ventricular hypertrophy on ECG. Values in mm (= 0.1 mV). Do not use code A:1 or A:2 in the presence of pre-excitation or complete left bundle branch block. Use the codes with precaution in the presence of myocardial infarction.

Code A:1		Women	Older men	Men < 35 years
R _{aVL}	≥	10	12	14
R _I	≥	16	18	20
R _{V₆} or S _{V₁}	≥	22	24	26
R _{V₅}	≥	28	30	32

Code A:2

If code A:1 is not present, but there is left axis deviation ($< 0^\circ$) or any of the Punsar codes I₁₋₅, reduce the values in code A:1 by 2 mm.

To construct criteria useful in clinical work, one has to reduce the number of items and make them easily memorable. Therefore, only five leads (aVL, I, V₁, V₆ and V₅) have been proposed in the present modification. Furthermore, by adjusting the limits to fit into an arithmetic series, they are also easy to remember (Table 5.III). The relationship between the arbitrary limits proposed and the upper limits, from which they are derived, is illustrated in Fig. 5.1. The leads chosen are those most commonly encountered in previous studies.

For general clinical use it was considered that a "falsely" positive diagnosis of LVH at a rate of 2.5 % of clinically healthy subjects with normal blood pressures was not acceptable. The suggested amplitude criteria for LVH was therefore set higher than the 97.5 % percentile. On the average it is 3.7 mm above the 97.5 % percentile.

It should be noted, that the inferior leads are not used. The reason for this is, that the maximal spatial vector shifts to a more superior direction with increasing blood pressure, thereby lowering the inferior amplitudes (McCaughan et al 1973). Inferior lead R-waves are therefore not likely to increase with left ventricular hypertrophy, at least not when this is caused by high blood pressure.

Amplitude criteria of left ventricular hypertrophy (LVH) are based on the fact that the myocardial mass increases, which leads to increased amplitudes. With advancing hypertrophy, however, fibrosis takes place (Ackerman 1950, McPhie 1958, Mazzoleni 1975). Fibrosis of the myocardium reduces the amplitudes (Weinberg 1950, Simonson 1961). It is therefore reasonable to assume that the deflection amplitudes will be reduced in hypertrophied hearts of long standing. Consequently, it is also reasonable to accept lower amplitudes as a sign of LVH if signs of fibrosis are also present in the ECG. Signs of fibrosis in an ECG are prolongation of the QRS-interval (Weinberg 1950, Mazzoleni 1975), left axis deviation (Corne 1965, Grant 1956) and ischemic ST-segment changes (Weinberg 1950).

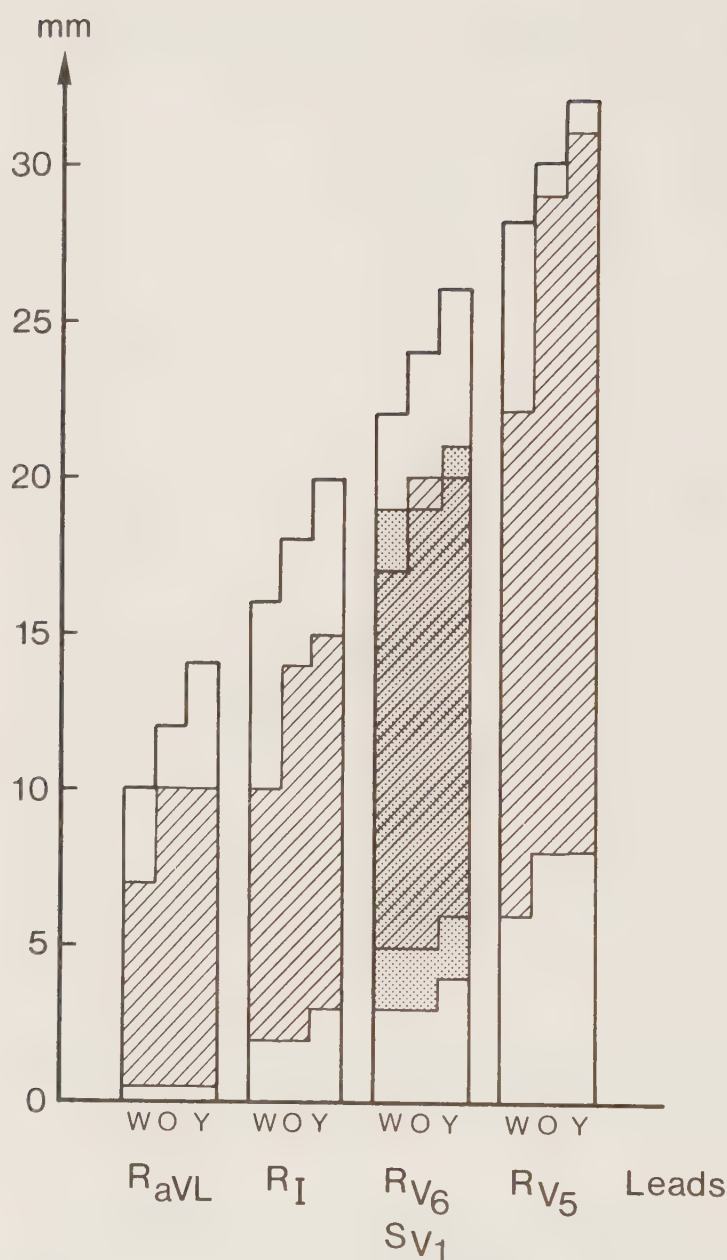


Fig. 5.1. The relationship between the limits for left ventricular hypertrophy according to the new Code A:1 (top of the bars) and the 95 % confidence interval for R-wave amplitudes in leads aVL , I , V_6 and V_5 (hatched areas) and S-wave amplitude in lead V_1 (dotted area). W = women, O = older men, Y = younger men (less than 35 years).

In the Framingham study (Kannel 1970) it was hypothesized that LVH with ST and T-wave abnormalities indicated ischemic myocardial involvement in the hypertrophied heart, representing a more advanced form of hypertrophy. Such "fibrotic hypertrophies" are easy to miss in a classification system where consideration is not taken to the reduction of amplitudes with fibrosis.

An additional code (A:2) has therefore been proposed in the present study. Code A:2 (Table 5.III) implies that the arbitrary limits of code A:1 are 2 mm lower in the presence of fibrotic signs in the ECG, i.e. left axis deviation (less than 0^0) or ischemic ST-segment changes (Punsar codes I₁₋₅). Prolongation of the QRS-interval was not judged to be a suitable indicator in this context.

Codes A:1 and A:2 were found in seven subjects (1.9 %) of the accepted sample. Code A:1 was found in two men and two women. One man, aged 32 years, had $SV_1 = 31$ mm. He was slender and smoked heavily. The other man, aged 26 years, was an active athlete and he had $RV_5 = 34$ mm. One woman, aged 32 years, had $RV_5 = 29$ mm. She was thin (weight 60 kg and height 175 cm) and was a heavy smoker. The other woman, aged 57 years, had an $R_{aVL} = 14$ mm and $RI = 17$ mm. She was an overweight (86 kg), smoking lady with a blood pressure of 155/95. Code A:2 was found in three men and they all had diastolic blood pressures in the upper range of the normal. One man, aged 31 years, had LAD, a QRS-duration of 0.11 s and an $R_{aVL} = 13$ mm (subject 1 in Fig. 3.2). (Six months after the investigation he was admitted to the hospital with anterolateral infarction.) His blood pressure was 135/90. One man, aged 43, had LAD and $R_{aVL} = 10$ mm. He was overweight and had a blood pressure of 150/90 (subject 4 in Fig. 3.2, ECG and VCG in Fig. 8.2). The third man, aged 51 years, was grossly overweight (weight 87 kg and height 166 cm) and he had a blood pressure of 145/95 (part of ECG reported in Fig. 2.8). He had $R_{aVL} = 10$ mm. His MFQRS-axis was 0^0 , and he qualified for the code A:2 by the presence of ischemic ST-segments.

5.2 VALIDATION OF THE NEW CRITERIA FOR LVH

The proposed modifications of Minnesota code items for R-wave amplitude, codes A:1 and A:2, were tested on non-averaged ECGs of men and women with high and low blood pressure. The male and female samples had both been studied previously by Thulin (1978) and by Thulin et al (1978). The age and blood pressure and number of subjects in the hypertensive and control groups are reported in Table 5.V. This table shows that the hypertensive men had a lower age and a lower systolic and diastolic blood pressure than the hypertensive women. The differences are statistically significant.

The number of ECGs codable according to the Minnesota codes 3.1 and 3.3 and to the new codes A:1 and A:2 are reported in Table 5.VI. For comparison, the results are also given for the point score system of Romhilt and Estes (1968). Electrocardiographic signs of LVH were most prevalent among the hypertensive women in all systems tested, as could be expected from the differences in blood pressure and age (Table 5.V).

The point score system of Romhilt and Estes (Table 5.VII) has been used for comparison, because it is possibly the best system for electrocardiographic diagnosis of left ventricular hypertrophy (Johnson et al 1977). Its sensitivity has been estimated to be 40-54 %, and its specificity to be 96-100 % (Romhilt et al 1969, Geva et al 1979). With this system, two men and six

women were scored for LVH (Table 5.VII). It is interesting to note that only one of the women was scored because of a high amplitude R-wave (lead II = 20 mm). The other five women were scored for LVH due to combinations of ST-T-changes, left atrial involvement, left axis deviation and QS-duration. The two men both had high amplitude deflections. Fourteen men were scored for LVH by the Minnesota code, and the majority of these were in the low blood pressure group. This suggests low specificity, especially for the Minnesota code 3.3. In women, however, significantly more subjects in the high blood pressure group were scored according to the Minnesota code for LVH than in the low blood pressure group.

Code A scored four men in the high blood pressure group and none in the low blood pressure group. Thirteen of the women in the high blood pressure group were scored for LVH compared to three in the low blood pressure group. The difference is significant ($\chi^2 = 6.75$, $p < 0.01$). Of the eight subjects, scored for LVH by the Romhilt and Estes' system, two women were not scored by Code A. One was the woman with the high R-wave amplitude in lead II, a lead which is not included in Code A. The other woman scored for LVH by ischemic ST-segment in lead V_6 and a QS-duration of 0.09 s (score 4).

It appears as if code A has better sensitivity than the point score system of Romhilt and Estes, and better specificity than the Minnesota code, at least in men. However, the precise sensitivity and specificity of Code A, has to be determined in larger clinical materials examined with preferably also other methods for the estimation of LVH.

Table 5.V. Age and blood pressure of men and women used to test the validity of the new code for left ventricular hypertrophy (Code A). The high blood pressure groups (HBP) consist of subjects from above the 95:th percentile of the blood pressure distribution. The age-matched controls (LBP) were from below the 30:th percentile. Students' test has been applied, and significance between the sexes is reported.

*** = $p < 0.01$, *** = $p < 0.001$, NS = not significant.*

	HBP			LBP		
	♂ n=31	Signifi- cance	♀ n=32	♂ n=31	Signifi- cance	♀ n=32
Age (years)	40 ± 10	**	48 ± 15	42 ± 11	NS	47 ± 13
Systolic blood pressure (mm Hg) M ± SD	146 ± 15	***	175 ± 34	117 ± 10	NS	117 ± 8
Diastolic blood pressure (mm Hg) M ± SD	93 ± 11	**	102 ± 13	72 ± 6	NS	73 ± 6

Table 5.VI. Number of subjects with high (HBP) and low (LBP) casual blood pressure, and whose ECGs had high amplitude items according to the point score system by Romhilt & Estes (1968), the Minnesota code and to a new code (code A), proposed in the present study. χ^2 test has been applied to the differences between HBP and LBP groups, the main code and the optional code being taken together. LVH = left ventricular hypertrophy.
 * = $p < 0.05$, ** = $p < 0.01$, NS = not significant.

	MEN			WOMEN		
	HBP n=31	Diff	LBP n=31	HBP n=32	Diff	LBP n=32
Romhilt & Estes						
Definite LVH (score 5)	2	NS	0	4	*	0
Possible LVH (score 4)	0		0	2		0
Minnesota code 3.1	4	NS	2	6	*	3
" " 3.3	1		7	5		1
Code A:1	3	NS	0	11	**	2
Code A:2	1		0	2		1

Finally it might be of interest to test the new code on the 27 subjects excluded from this study because of high blood pressure. Mean values of age, blood pressure, electrical axis, obesity index and relevant R-wave amplitudes are reported in Table 5.IV. The table shows an axis deviation to the left in the subjects with high blood pressure, especially in women. The greater obesity index in subjects with high blood pressure may contribute to the axis deviation to the left. The R-wave amplitudes change accordingly, i.e. the "hypertensive" subjects have higher amplitudes in aVL and I and -aVR, but lower amplitudes in II, aVF and III than the normotensive subjects. There were no differences of precordial amplitudes between "hypertensive" and normotensive subjects. Hypertensive women were older than the controls.

The code A:1 was found in only one woman with $R_{aVL} = 12$ mm and $R_I = 17$ mm. She was 50 years old and undergoing treatment for hypertension. The code A:2 was found in only one man, aged 38, with a blood pressure of 150/100. Three other men were codable according to the Minnesota codes 3.1 or 3.3. LVH according to Romhilt and Estes was not found in either men or women.

The fact that the new code A was uncommon among those 27 subjects with elevated blood pressure is surprising. Although the blood pressure were casual, one can except some of the subjects to be hypertensive in the clinical sense. There are three possible explanations for the unexpected low prevalence of code A. Firstly, the subjects with elevated blood pressure were overweight compared to subjects of the accepted sample. It has been shown by Pipberger et al (1967) that overweight reduces the magnitude of the spatial vectors. This reduction of absolute amplitude counteracts the increase of R-wave amplitudes in aVL and I, which would

6 REPOLARIZATION DEFLECTIONS

Repolarization of the myocardium is affected by a large variety of factors. Some of these, e.g. ischemia and electrolyte disturbances, are quite frequently seen in clinical practice. Unfortunately, the repolarization phase is also affected by factors operating in the normal individual, e.g. activation of the sympathetic nervous system by anxiety. Regardless of their causes, the ST- and T-changes are of similar appearance.

The present study contains a detailed quantitative and qualitative analysis of ST and T. For convenience the results for ST and T are separately presented and discussed. The ST-segment is described in terms of amplitude of ST-junction and inclination of the segment. These features will be presented first. Of special interest are the horizontal or downward sloping ST-segments (HoDST), and their prevalence has therefore been analysed in the presence or absence of ST-junction depression. Finally, a detailed classification according to Punsar et al (1968) has been performed in leads V₄, V₅ and V₆. The Punsar code considers not only HoDST, but also ascending ST-segments with ST-junction depression.

6.1 THE ST-JUNCTION AMPLITUDE

The ST-junction is the usually well-defined transition from QRS to ST. This junction (J) is thought to represent the end of depolarization. It is occasionally difficult to find the J-point, as the transition from QRS to ST is gradual.

The amplitude of the ST-junction was found to vary with sex, age and position of the heart. The variations with age and position were small and without practical importance when delineating normal limits. The sex differences, however, were large and they are reported in Table 6.I.

Significant sex differences are noted in leads -aVR, II and aVF, and in V₁-V₄. The largest amplitudes are found in leads V₂-V₃, where the upper limit in men is 2 mm and in women 1 mm.

The normal limits are illustrated in Fig. 6.1. In the limb leads, it can be noted that elevations and depressions of the ST-junction generally are small. In the precordial leads, however, not only the largest elevations are observed, but also the deepest depressions.

An ST-junction depression of 0.5 mm or more but less than 1 mm qualifies the ECG to either of codes I₃, S₃ or R₃ according to Punsar et al (1968) (Fig. 2.5). In leads V₄ (men and women) and V₅ (women) such ST-junction depressions were found so commonly that they occur within the normal limits. In lead V₄ 12 out of 16 cases had R₃ and four S₃. In lead V₅ eight out of nine women had S₃ and one R₃. It seems reasonable to accept code R₃ as normal in lead V₄ in men and women and to accept code S₃ as normal in lead V₅ in women.

Table 6.1. ST-junction amplitude (mm) in men and women. 1 mm = 0.1 mV. UL = 97.5 % percentile. LL = 2.5 % percentile. The significance of the differences between the sexes (Student's *t*-test) is indicated by: *** = $p < 0.001$, ** = $p < 0.01$, NS = not significant.

Sex	Lead	Lead											
		aVL	I	-aVR	II	aVF	III	V ₁	V ₂	V ₃	V ₄	V ₅	V ₆
MEN n=163	Mean	0	0	0.1	0.1	0.1	0	0.3	0.9	0.6	0.2	0	0
	SEM	0.1	.01	.02	.02	.02	.01	.02	.04	.04	.04	.02	.02
	UL	0.3	0.4	0.5	0.7	0.7	0.5	1.1	2.1	1.9	1.2	0.7	0.6
	LL	-0.3	-0.4	-0.3	-0.4	-0.3	-0.3	-0.1	0	-0.1	-0.6	-0.4	-0.4
		NS	NS	***	***	***	NS	***	***	***	***	NS	NS
WOMEN n=194	Mean	0	0	0	0	0	0	0.1	0.4	0.1	0	0	0
	SEM	.01	.01	.01	.02	.01	.01	.02	.03	.02	.02	.01	.01
	UL	0.2	0.2	0.4	0.4	0.4	0.4	0.6	1.1	1.0	0.6	0.3	0.3
	LL	-0.3	-0.3	-0.3	-0.5	-0.4	-0.4	-0.3	-0.2	-0.5	-0.7	-0.6	-0.4

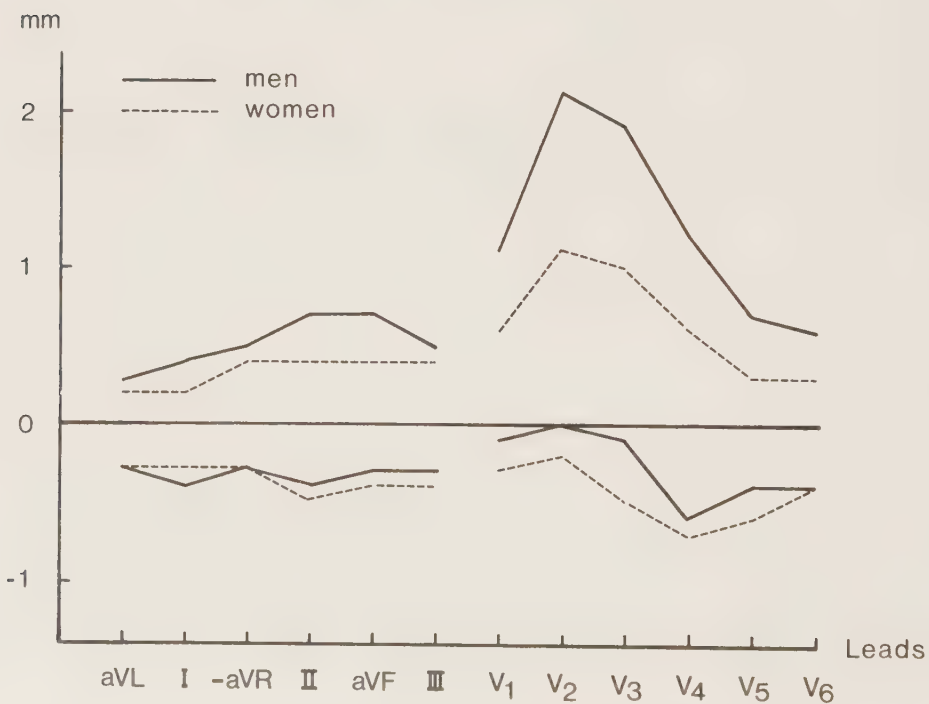


Fig. 6.1. Normal limits of ST-junction amplitude (mm) in men and women. Upper and lower limits are given as the 2.5 % and 97.5 % percentiles. 1 mm = 0.1 mV.

6.2 THE ST-SEGMENT INCLINATION

a) The slope

The ST-segment inclination has been calculated in all 12 standard leads (see Methods, Fig. 2.3). Large inter-observer variations make manual methods impractical in clinical work. Normal limits will therefore not be reported. The results will only be presented as mean values in sex and age groups. However, the inclination is a suitable quantitative measure and can be used in studies with a limited number of specially trained observers.

Mean values of ST-segment inclination in men of different age groups are reported in Appendix No. 5 and plotted in Fig. 6.2. *In men, the ST-segment inclination decreased with age in all leads, except lead III, as seen at analysis of the variance (F-test).* The steepest inclination was found in leads V_2 - V_4 , and there was a progressive change of inclination between the leads.

The ST-segment inclination in women is reported in Appendix No. 5. *In women the inclination decreased with age in leads aVL-II and V_4 - V_6 (F-test).* In V_1 , there was an increase with age, which is in contrast to men. This apparently paradoxal pattern may be explained by a high frequency of horizontal and downward sloping ST-segments in younger women in lead V_1 . In leads aVF, III and V_2 - V_3 , no age variation was seen. The steepest inclinations were seen in V_2 - V_4 as in men, and there was a progressive change of inclination between the leads.

Mean values of the inclination were always positive and the steepest slopes were found in men. These *sex differences* were significant in all leads except lead aVL (Table 6.II). The sex difference in young subjects is illustrated by the ECGs in Fig. 2.1 and 6.3. The difference levels off with age, but is still significant in leads I, -aVR and V_1 - V_6 in age group 45-60 years (Table 6.III). Thus, sex differences in older subjects were more wide spread for ST-segment inclination than for R-wave amplitude, where significant differences were found only in leads V_4 and V_5 in age group 45-60 years (Table 4.X).

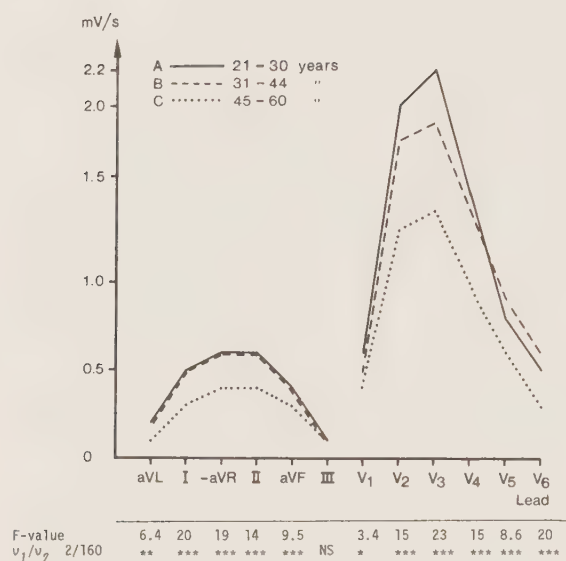


Fig. 6.2. Mean values of ST-segment inclination (mV/s) in men in three age groups, A, B and C. The values are plotted from the Appendix No. 2. The significance of differences between age groups is reported as the F-value. * = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$ v_1 and v_2 = degrees of freedom.

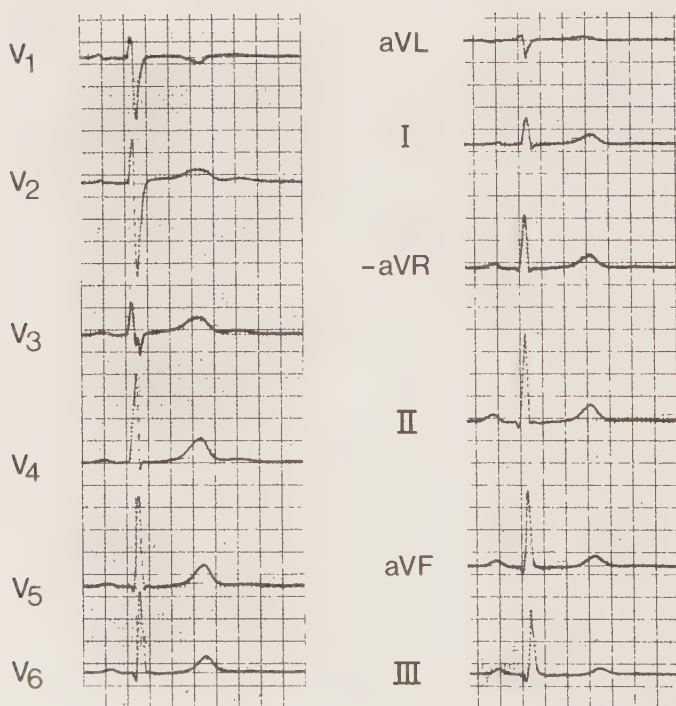


Fig. 6.3. ECG from a young healthy woman, aged 25 years. Note the slow ST-segment inclination giving the T-waves a symmetrical appearance. Also note the negative T-wave in lead V_1 , a feature which is very common in women.

Table 6.II. ST-segment inclination (mV/s) in men and women, aged 21-60 years. The sex differences were evaluated with Student's *t*-test.

** = $p < 0.01$, *** = $p < 0.001$, NS = not significant.

		aVL	I	-aVR	II	aVF	III	V ₁	V ₂	V ₃	V ₄	V ₅	V ₆
MEN	M	.19	.45	.52	.56	.34	.08	.49	1.74	1.86	1.35	.78	.46
n=163	SEM	.015	.017	.022	.022	.020	.021	.032	.059	.054	.042	.031	.021
Difference		NS	***	***	***	***	**	***	***	***	***	***	***
WOMEN	M	.16	.31	.36	.38	.21	.01	.17	.88	.88	.62	.42	.27
n=194	SEM	.010	.013	.013	.015	.013	.013	.019	.032	.027	.022	.016	.013

Table 6.III. ST-segment inclination (mV/s) in men and women, aged 45-60 years. The sex differences were evaluated with Student's *t*-test.

* = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$, NS = not significant.

		aVL	I	-aVR	II	aVF	III	V ₁	V ₂	V ₃	V ₄	V ₅	V ₆
MEN	M	.14	.34	.38	.39	.25	.06	.41	1.32	1.43	1.01	.57	.30
n=46	SEM	.027	.030	.029	.032	.029	.035	.043	.080	.075	.068	.057	.043
Difference		NS	**	*	NS	NS	NS	**	***	***	***	**	*
WOMEN	M	.12	.24	.31	.33	.19	.02	.25	.85	.86	.58	.39	.20
n=48	SEM	.016	.020	.020	.024	.022	.020	.039	.053	.060	.052	.027	.022

b) Prevalence of horizontal or downward sloping ST-segments

The horizontal or downward sloping ST-segments (HoDST) are of special interest as they are related to heart disease (Blackburn et al 1960, Punsar et al 1968, The Pooling Project 1978). This section contains an analysis of HoDST in all twelve leads (Table 6.IV). In section 6.3 a more detailed analysis of ST in V₄-V₆ will be presented. The presence or absence of ST-junction depression concomitant to HoDST was noted. To avoid coding of ST in the presence of negative T-waves in right ventricular leads, ST-segment configuration was considered only when the preceding QRS-complex had an R/S-ratio > 1.

The prevalence of ECGs with HoDST was 5.3 % (5 men, 14 women) when only leads I, II -aVR and V₁-V₆ were considered. The prevalence of HoDST rose to 12 % with lead aVL and to 16 % (20 men, 36 women) if lead aVF was also included.

HoDST with ST-junction zero or depressed was 3.1 % in men (5/163) and 5.2 % in women (10/194), if only leads I, II, -aVR and V₁-V₂ were considered. With aVL included, the prevalence rose to 6.8 % in men and to 11 % in women. With aVF, the prevalence rose to 9.2 % in men and 13 % in women. The ST-junction depression was in all instances less than 0.5 mm, i.e. codable according to Punsar code I₄ or I₅.

It has been pointed out by Punsar (personal communication 1983) that the prognostic significance of ST-segment changes differs according to in which lead(s) that they occur. From the results of this study, it seems for example, likely that the high prevalence of HoDST in leads aVL, aVF and III (Table 6.IV) does not reflect heart disease.

The location of the ST-junction in relation to the PQ-segment has been noted. HoDSTs are codable according to the classification system, introduced by Punsar et al (1968). Lead III is not included in the Punsar code but aVL and aVF are. In view of the high prevalence of HoDST in aVL and aVF in the present study, it appears reasonable to accept codes I₄ and I₅ as normal in these leads. The Punsar codes describe ST-segment depressions. In principle codes I₄ and I₅ may be associated with some ST-junction elevation notably, if the main part of the ST-segment is below the baseline. No definite rules have been described as how to regard various ST-patterns with ST-junction elevation. Werner et al (1976) suggested a special code, I₆, for downward sloping ST-segments in combination with ST-junction elevation. In the present study codes I₄ and I₅ do not include ECGs with ST-junction elevation. HoDST with ST-junction elevation was not uncommon in lead III (Table 6.IV).

However, it is possible that HoDST also with ST-junction > 0 have clinical significance (Evans 1952 and 1965). It is therefore important to recognize and classify them. Fig. 6.4 shows an ECG with such a change. It is suggested that not only downward sloping but also horizontal ST-segments with ST-junction elevated (but not more than 0.5 mm) should be classified as I₆ and added to the Punsar code.

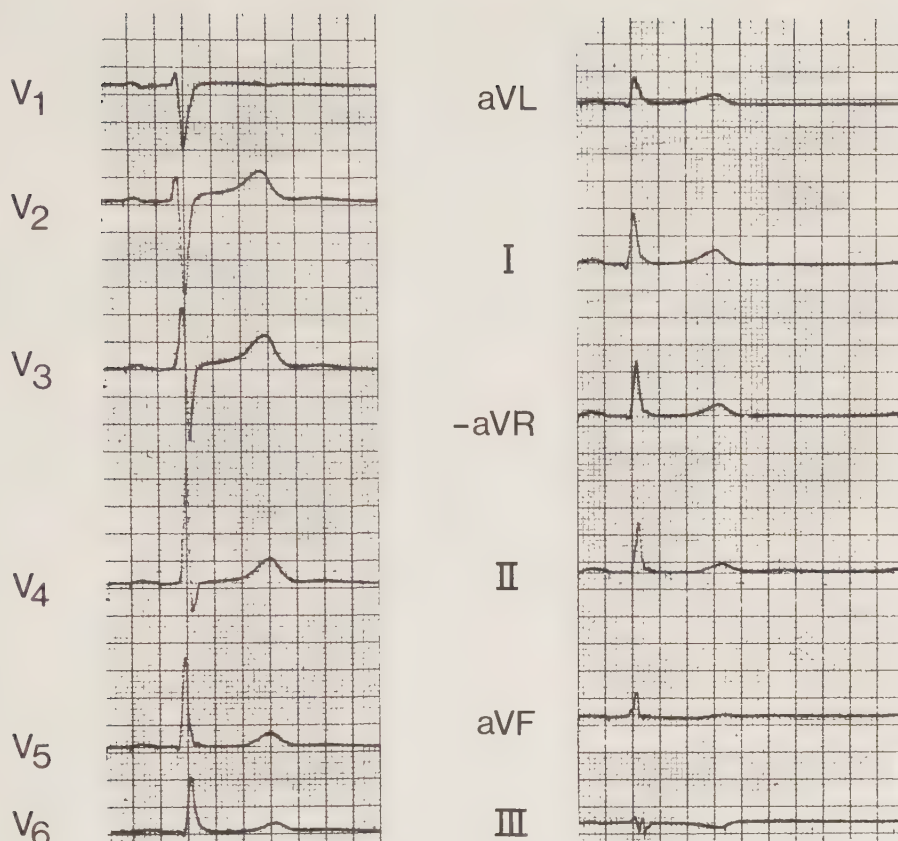


Fig. 6.4. ECG of a man aged 53 years. The flat ST-segment in lead V₅ is not codable according to the Punsar system (1968), as the ST-segment is not depressed compared to the PQ-segment. It is, however, reasonable that it should influence the clinical judgement of this ECG. The ST-segment in V₅ is coded 4.4.2 according to the new standard presented in chapter 9. The ST-segment in aVF is coded as I5 according to Punsar.

Table 6.IV. Number of horizontal or downward sloping ST-segments (HoDST) in eight standard leads. In each lead, the ST-segment configuration was considered only when the QRS-complex had an R/S > 1. The ST-segments are grouped according to the presence or absence of ST-junction (STJ) depression. HoDST-segments were not found in leads V_1 - V_3 or $-aVR$, when R/S-ratio > 1.

		aVL	I	II	aVF	III	V_4	V_5	V_6
MEN n=163	STJ > 0	3			2	14		1	
	STJ $\leq$ 0	6	1	2	6	29		1	3
	Sum	9	1	2	8	43	0	2	3
WOMEN n=194	STJ > 0	3		1	5	18		2	1
	STJ $\leq$ 0	11		7	14	51	2	3	4
	Sum	14	0	8	19	69	2	5	6

6.3 CLASSIFICATION OF THE ST-SEGMENT

Classification or "coding" of the ST-segment has been done according to the system proposed in 1968 by Punsar et al (Fig. 2.5). Consideration is taken not only to horizontal or downward sloping ST-segments but also to ascending ST-segments. The coding was performed in leads V_4 , V_5 and V_6 , both by a computer and by two independent observers. If there were inter-observer or observer-computer disagreements a third observer decided the final code (see Methods, 2.5).

The Punsar code considers only leads in which the ST-segment is depressed. Depending on the inclination, the ST-segments are classified into either of three classes. The most serious class is the ischemic (I) with horizontal or downward sloping ST-segment. Then follows the class with slowly ascending ST-segments (S) and the least serious class with rapidly ascending ST-segments (R).

Punsar codes were found in 11 % of the accepted sample. A code was more often found in women (31/194 = 16 %) than in men (10/163 = 6 %). The difference is significant ($\chi^2 = 7.50$, $p < 0.01$) (Table 6.V). The codes found were of the ischemic type (I4-5), slowly ascending (S3-4) as well as rapidly ascending (R3). The significant sex difference was due to a higher frequency of slowly ascending ST-segments in women. Thus, slowly ascending ST-segments were found in 12 % of the women compared to 2 % in the men. There was no significant sex difference for the ischemic or the rapidly ascending categories.

Age variation is shown in Table 6.VI. Age variation in men was significant if the ischemic and slowly ascending categories were grouped together ($\chi^2 = 4.70$, $p < 0.05$). There was no age variation in women.

The distribution of the Punsar codes in the three leads V4-V6 is illustrated in Table 6.VII. In men, ischemic and slowly ascending ST-segments were not found in lead V4, and no rapidly ascending ST-segments in leads V5-V6. In other words, the more serious codes are located to the left on the precordium. The same trend is present among women, although ischemic (I5) and slowly ascending (S3-S4) ST-segments are seen in V4.

Table 6.V. The ST-segment in men and women. Number of ECGs of the accepted sample (n=357) with codable items according to the Punsar code in one or more of leads V4-V6. ECGs were classified according to the most severe code found.

Punsar code	MEN n=163	WOMEN n=194	chi ²
I ₄₋₅	3	7	NS
S ₃₋₄	4	23	9.89**
R ₃	3	1	NS
Sum	10	31	7.50**

Table 6.VI. The ST-segment and age. Number of ECGs with ST-segment codable according to Punsar in V4-V6. ECGs are classified according to the most severe code found. Grouping according to sex and age has been made. A = 21-30 years, B = 31-44 years, C = 45-60 years.

		MEN			WOMEN		
Punsar code \ Age n		A 66	B 51	C 46	A 83	B 63	C 48
I ₄			1	2			
I ₅					5	1	1
S ₃		1			2	4	5
S ₄				3	4	4	4
R ₃		1	1	1			

Table 6.VII. Number of items according to the Punsar code in leads V_4 - V_6 . The items are grouped in the three categories, ischemic (I), slowly ascending (S) and rapidly ascending (R).

Punsar code	MEN, n = 163			WOMEN, n = 194		
	V_4	V_5	V_6	V_4	V_5	V_6
I		1	3	2	3	4
S		5	3	4	20	15
R	6			6	1	
Sum	6	6	6	12	24	19

6.4 THE T-WAVE

a) The T-wave amplitude

The T-wave amplitude was found to vary with sex, age and position of the heart. Variation with position of the heart was small, and of no practical importance when the normal limits were delineated.

Statistically significant differences between mean values of different age groups were present in most leads, especially in men (in all leads except aVL, I and V_1), but also in women (in all leads except aVF, III and V_2 - V_4). T-wave amplitudes decreased with age. However, when the 2.5 % and 97.5 % percentiles were considered, the differences were small. In other words, high amplitude T-waves were not uncommon among older subjects, and low T-waves were not uncommon among the young subjects.

The sexes differed significantly not only as concerns mean values of T-wave amplitude in all leads except aVL, but also with regard to the normal limits as seen in Table 6.VIII. This is a matter that appears to be of importance in clinical evaluation of ECGs.

The T-wave amplitude was in general larger in men than in women (Table 6.VIII). In Fig. 6.5 the upper and lower limits in men and women have been graphically illustrated. The highest amplitudes were found in the precordial leads V_2 - V_4 and in the limb leads -aVR and II. Inverted T-waves were frequent in leads aVL, III and V_1 . Inverted T-waves in V_1 were especially frequent, being present in 86 women (44 %) and 18 men (11 %). The sex difference is significant ($\chi^2 = 45.9$, $p < 0.001$). The ST-junction was depressed in two of the men and twelve of the women. However, the T-wave inversion were in most instances very small, and only in two men and five women did it exceed 2 mm. Persistent juvenile pattern refers to T-wave inversion in the right precordial leads. T-wave inversion in V_1 and V_2 was seen only in one woman. In all other instances the T-wave inversion was restricted to lead V_1 .

Table 6.VIII. T-wave amplitude (mm) in men and women. 1 mm = 0.1 mV.
 UL = upper limit (97.5 % percentile), LL = lower limit (2.5 % percentile).
 Sex differences tested with Students' t-test. * = $p < 0.05$, ** = $p < 0.01$
 and *** = $p < 0.001$.

Sex	Lead	aVL	I	-aVR	II	aVF	III	V ₁	V ₂	V ₃	V ₄	V ₅	V ₆
MEN n=163	Mean	0.8	2.2	2.7	3.2	2.1	1.0	1.7	7.2	7.4	6.0	4.1	2.9
	SEM	.064	.074	.073	.091	.082	.086	.12	.20	.20	.20	.15	.098
	SD	.82	.94	.93	1.2	1.0	1.1	1.6	2.6	2.6	2.5	1.9	1.2
	UL	2.4	4.4	5.0	6.3	4.4	3.2	4.9	12.8	13.4	12.6	9.2	5.8
	LL	-0.9	0.9	1.0	0.8	0.2	-1.1	-1.1	2.7	2.9	2.1	0.9	0.5
	Difference	NS	*	**	***	***	***	***	***	***	***	***	***
WOMEN n=194	Mean	0.9	2.0	2.4	2.7	1.7	0.6	0.2	4.1	4.5	3.8	3.0	2.3
	SEM	.045	.057	.054	.071	.059	.066	.083	.12	.13	.11	.088	.071
	SD	.63	.80	.75	1.0	.82	.91	1.1	1.7	1.8	1.6	1.2	1.0
	UL	2.2	3.8	3.9	4.8	3.3	2.3	2.7	7.7	8.7	7.8	5.7	4.4
	LL	-0.2	0.7	1.1	1.0	0.1	-1.1	-2.0	1.1	1.0	1.0	1.0	0.8
	Difference	NS	*	**	***	***	***	***	***	***	***	***	***

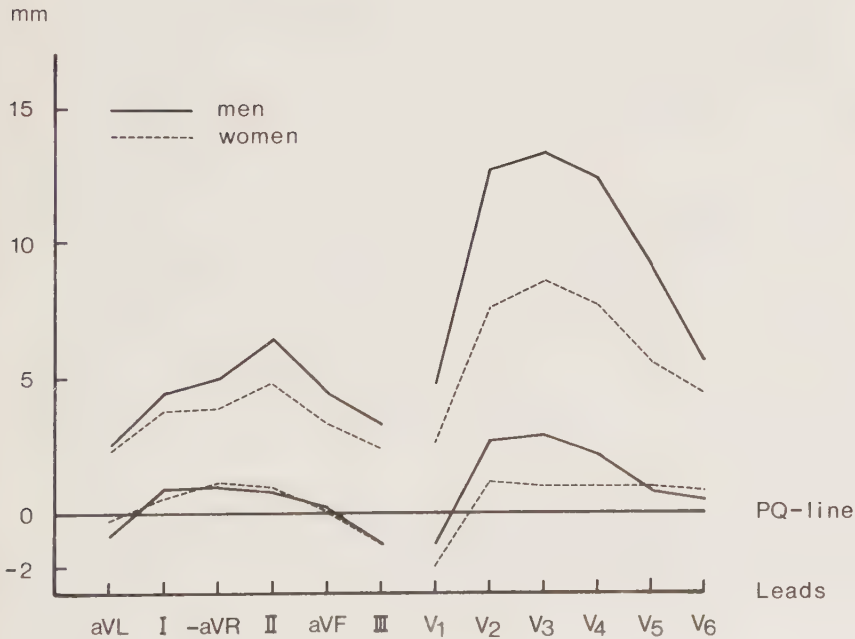


Fig. 6.5. Normal limits of the T-wave amplitude (mm) in 163 men and 194 women. Lower and upper limits correspond to the 2.5 % percentile and the 97.5 % percentile, respectively, from Table 6.VIII.

b) The T/R-ratio

The T/R-amplitude ratio has been calculated for all subjects in all twelve leads, and the results are reported in Table 6.IX. Statistically significant sex differences were found in leads -aVR, II, aVF and V_1 - V_4 with larger ratios in men than in women.

Table 6.IX. The T/R-amplitude ratio in men and women. Mean values, standard deviation (SD) and upper (UL) and lower limits (LL) i.e. the 97.5 % and 2.5 % percentiles, respectively, are reported. Sex differences were evaluated with Students' t-test and t-values are reported.

* = $p < 0.05$, ** = $p < 0.01$ and *** = $p < 0.001$, NS = not significant.

SEX	LEAD	aVL	I	-aVR	II	aVF	III	V_1	V_2	V_3	V_4	V_5	V_6
Men n=163	Mean	.41	.43	.35	.33	.36	.17	.62	1.31	.93	.40	.27	.25
	SD	.59	.23	.15	.18	.61	.37	.76	1.07	.69	.22	.13	.13
	UL	2.0	1.1	0.8	0.8	1.0	1.0	2.8	4.0	2.9	1.1	0.6	0.6
	LL	0.4	0.1	0.1	0.1	0.02	-0.6	-0.5	0.2	0.1	0.08	0.06	0.06
Women n=194	Mean	.55	.39	.32	.28	.24	.14	0.009	.90	.68	.32	.26	.24
	SD	1.22	.25	.13	.13	.18	.40	1.09	.69	.42	.15	.11	.10
	UL	2.0	1.0	0.7	0.6	0.8	1.1	2.0	3.0	1.8	.8	.5	.5
	LL	-0.2	0.1	0.1	0.1	0.02	-0.5	-1.2	0.2	0.1	0.08	0.08	0.08
Students' t-value		NS	NS	*	***	**	NS	***	***	**	**	NS	NS
		-1.33	1.56	2.03	3.09	2.60	0.73	6.02	4.35	2.99	4.07	0.79	0.82

6.5 CLASSIFICATION OF THE T-WAVE

The T-waves have also been classified according to the Minnesota code V.1-4, here called 5.1-5.4 (Table 6.X). Codes 5.1 and 5.2 were not found but codes 5.3 and 5.4 were found in eleven subjects (7 men, 4 women).

T-wave code 5.3 was found in six men and two women. In clinical practice, the T-wave is often evaluated in relation to the R-wave as in the optional code 5.4. In this study, only one man and two women fulfilled the criteria for code 5.4.

The only T-wave inversion not included in the Minnesota code is a terminal dip of the T-wave less than 1 mm. This was found in 10 men, but in no woman in the present study. Five of the men had the item in V_1 only. In all other leads the item was rare. A terminal dip of the T-wave has been regarded as one of the smaller signs of ischemic heart disease (Evans 1952).

The overall prevalence of T-wave items 5.3-4 was 3.1 %. Although not statistically significant, the prevalence was higher in men (4.3 %) compared to women (2.1 %).

Analysis of the VCG of the eleven subjects with a Minnesota T-wave item showed pathological QRS-loop in two men (postero-inferior infarction in one and infero-apical infarction in one).

The T/R-ratio criteria in Minnesota code 5.4 is less than 0.05. In the literature (Short 1972, McPhie 1958, Sokolow and Friedlander 1949), a T/R-ratio of less than 0.1 in corresponding leads has been suspected to indicate early ischemic changes. The prevalence of this item in the present study was high. Under the age of thirty-five, for example, 18 % of the women (19/108) had the item, compared to 9 % (8/89) of the men (leads I, II, aVF, V₃-V₆). This item therefore appears to be of little clinical value.

Table 6.X. *The Minnesota code for T-wave items (Rose 1968).*

T-wave items

(Measured from following baseline level at onset of P; do not code in presence of ventricular conduction defects 6.4, 7.1, 7.2, 7.4.)

- 5.1 T-amplitude negative, minus 5 mm or more in any of leads I, II, V₂, V₃, V₄, V₅, V₆ or in lead aVL when R-amplitude is 5 mm or more, or in lead aVF when QRS is mainly upright.
 - 5.2 T-amplitude negative or diphasic (positive-negative or negative-positive type) with negative phase at least minus 1.0 mm but not as deep as minus 5 mm in any of leads I, II, V₂, V₃, V₄, V₅, V₆, or in lead aVL, when R-amplitude is 5 mm or more, or in lead aVF when QRS is mainly upright.
 - 5.3 T-amplitude zero (flat), or negative, or diphasic (negative-positive type) with less than 1.0 mm negative phase in any of leads I, II, V₃, V₄, V₅, V₆, or in lead aVL when R-amplitude is 5 mm or more; not coded in lead aVF.
 - 5.4 (Optional code): T-amplitude positive and T/R-amplitude ratio less than 1/20 in any of leads I, II, aVL, V₃, V₄, V₅, V₆; R-wave amplitude must be 10 mm or more.
-

7 DISCUSSION

7.1 THE ELECTRICAL POSITION OF THE HEART

The present study shows that the mean frontal QRS-axis (MFQRS-axis) in men shifts to the left with age. This is in line with most other studies. The prevalence of the Minnesota code 2.1 (MFQRS-axis $< -30^\circ$), here called extreme left axis deviation (ELAD) increases with age in men (Blackburn 1967, Ostrander et al 1965, Hiss et al 1960 and 1962, Goldberg et al 1970, Jonsson 1977, Waern 1978, Durakovic et al 1981, Östör et al 1981). Also when mean values are studied, the leftward shift with age is evident (Hiss et al 1960, Simonson 1961 and 1972, Blackburn et al 1967). The age variation appears to level off after the age of 40 years (Hiss et al 1960, Simonson 1961 and 1972, Blackburn et al 1967). The increasing prevalence with age of ELAD in population studies is in contrast to the fact that the age variation of the mean values levels off after the age of forty. A possible explanation is that ELAD is a sign of heart disease and consequently more common among older subjects in unscreened population studies, in which the prevalence of heart disease increases with age. In studies of healthy populations, these subjects are excluded to the extent that the heart disease is detected. Such a selection of course influences the mean values, especially in age groups where disease is common, i.e. old age. This could explain why age variation of the mean values levels off with age in healthy populations.

The present study indicates that variation of the MFQRS-axis with age is less pronounced in women. This is in contrast to other studies, where an increase of the prevalence of Minnesota code 2.1 has been observed with age (Ostrander 1967, Goldberg 1970, Jonsson 1977, Durakovic 1981, Östör 1981). Also when mean values are studied, there is a reduction of the axis with age. Furthermore, the relationship seems to be linear up to the age of 50 years (Simonson 1961). Differing age variations in men and women may be explained by contributing factors, such as body build and blood pressure. In the present study, it was found that obesity (expressed as obesity index) and diastolic blood pressure influenced the MFQRS-axis as age did. This has also been shown in other studies (Simonson 1961, Rautaharju et al 1961). In the women of the present study, diastolic blood pressure had only a small influence on the MFQRS-axis. Obesity had a larger influence, but on the other hand obesity did not change with age in women, young women having the same obesity index as old women. It is interesting to see that after correction for obesity the MFQRS-axis did not vary with diastolic blood pressure (Appendix No. 1). Varying obesity in other studies in women may be the cause of MFQRS-axis dependence on age. Also, it is possible that the age variation is significant in men because of a joint effect of obesity and blood pressure as these factors increase with age.

The present study shows that the MFQRS-axis is influenced by body build. Heavy subjects (men and women) with a wide chest and a large obesity index are likely to have a MFQRS-axis to the left probably representing a anatomically horizontal heart position. This is in line with the studies by Simonson (1961) who found that the relative body weight reinforced the effects of age on the MFQRS-axis in men as well as in women. In the present study, however, obesity does not vary with age in women, which means that the influence of obesity on the MFQRS-axis does not reinforce age variation in women.

The present study indicated that LAD (MFQRS $< 0^\circ$) appears more common in men than in women. LAD was found in five men compared to one woman. This is in line with studies of the prevalence of the Minnesota code 2.1 (Seriki et al 1966, Ostrander et al 1967, Goldberg et al 1970, Jonsson et al 1977, Durakovic et al 1981, Östör et al 1981). ELAD was about twice as common in men compared to women in these studies. This is also reflected in the studies by Simonson (1961) who found the 97.5 % percentile to be more leftward for older men (-30°) than for older women (-15°). The corresponding values in the present study were -15° for older men and $+15^\circ$ for older women (Table 3.1). The differences may partly be explained by different selection procedures. Men and women in the Simonson study represented the working population compared to the randomly selected sample used in the present study. Also, the age distribution among men was different in the two studies. In the study by Simonson, older men (40-60 years) were twice as common as younger men (20-40 years) while in the present study, there were more younger men than older ones. Finally, it is also possible that the screening for disease was done in different ways in the two studies.

Furthermore, studies of the VCGs in the present study indicate that the left limit of the MFQRS-axis should be moved to 0° instead of -15° .

In other words, the present study indicated that LAD (MFQRS-axis $< 0^\circ$) is not only an aberrant ECG-finding but also a sign of disease. Five men (3.1 %) and one woman (0.5 %) were found to have LAD. Of these, three men had signs of subclinical IHD on VCG, another man was admitted with infarction 6 months after the investigation, and the woman had three risk factors for IHD, namely overweight, smoking and blood pressure in the upper normal range. It may seem odd to find so many subjects with signs of heart disease in a healthy population like the present one, but silent heart disease is probably not uncommon, not even in young physically fit men (Enos et al 1953, Mason 1963, Medalie 1976).

Extreme left axis deviation (ELAD = MFQRS-axis $\leq -30^\circ$) is rare in younger healthy subjects (Hiss et al 1962, Atterhög et al 1980) as well as in older healthy subjects (Gorman et al 1964, Soffer 1977). In population studies, where screening for disease is less thorough or not done, ELAD increases with age (Ostrander et al 1965, Goldberg et al 1970, Jonsson et al 1977, Waern 1978, Östör et al 1981, Durakovic et al 1981). Age trends of ECG-features in asymptomatic populations are to a large extent due to latent coronary artery disease (Simonson 1972).

Increased mortality for subjects with LAD has been shown especially for men aged 40-60 years (Östör et al 1981, Blackburn et al 1970, Rabkin et al 1981). On the other hand, subjects younger than 40, or older than 60 years, did not run a higher mortality risk than subjects with normal MFQRS-axis (Rabkin et al 1981). This may reflect different etiology of ELAD in different ages, such as myocarditis or congenital defects in the conductive system in the young subjects and fibrosis or without infarction in the elderly. Although the prognosis is better in these subjects, their LAD is likely to stem from an abnormal heart condition. Prognosis may also vary with different populations as shown in the study by Blackburn et al (1967). They found high prevalence of Minnesota code 2.1 in Greece and Italy, countries with low prevalence of IHD. Although LAD in some instances may have a good prognosis, it can be stated that an individual with LAD is more likely to be sick than healthy. Hence, it is reasonable to consider LAD (MFQRS-axis $< 0^\circ$), not only as an abnormal ECG-finding but also to be a sign of disease.

One must, however, consider that LAD must not always be due to abnormal electrical ventricular activity, but that it may be due to an unusual position of the heart. Goldberger (1953) focused attention on the fact that extreme CW-rotation (seen from below) in the horizontal plane often is observed together with ELAD in the frontal plane. This relationship demonstrates the importance of also considering the position in the horizontal plane when evaluating the position of the heart in the frontal plane, as was also stressed by Simonson (1961).

In this context it is interesting to consider the present study. The only man (subject No. 2) with LAD without signs of subclinical IHD according to VCG had an MFQRS-axis of -45° , the transitional zone in V₄ and an unusual Q-wave pattern with Q-waves only in leads aVL and I, and the absence of precordial Q-waves (Fig. 7.1). Such a pattern may indicate an extreme CCW-rotation around the long axis of the septum in a horizontal heart, combined with CW-rotation around the long axis of the body (seen from below in the horizontal plane), and backward rotation of the apex in the sagittal plane. To comprehend such a complicated rotation it is recommended to use Fig. 4.2, and imagine the position of the septal null plane with Q-waves only showing up in leads aVL and I.

One will find an inverted position of the heart, with the left ventricle positioned superiorly to the right ventricle. Once this is understood it is evident why this subject demonstrates ELAD. However, if the Q-wave pattern is ignored it would be more difficult to relate the ELAD in this patient to an unusual electrical heart position.

The present study introduces the concept of Q-axis as an alternative way to denote the electrical position of the heart. The direction of the Q-axis is opposite that of the septal vector. The septal vector results in the Q-wave in leads facing the left side of the septum. This has been recognised since the early days of ECG, and Goldberger (1953), among others have used the Q-wave as a marker for left ventricular leads. Unfortunately, previous systems of classifying the electrical position of the heart have resulted in too many positional groups to be practical for clinical work. To the best of my knowledge no one has previously analysed the Q-wave pattern in the same way as has been done in the present study. A possible reason may be that the "Cabrera system" for presentation of the limb leads (White 1971) has not been extensively used. The Cabrera system, used in the present study, facilitates detection of deflection patterns in the frontal plane.

The use of the Q-wave or Q-axis as markers for the electrical position of the heart has theoretical merits compared to the use of the mean QRS-axis. Durrer et al (1970) studied the total excitation of the isolated human heart. They found a consistent pattern of excitation during the first 10-15 ms after the start. Three endocardial areas were synchronously excited 0-5 ms after the start. The areas were all in the left ventricle, namely a) high on the anterior "paraseptal" wall just below the attachment of the mitral valve down toward the anterior papillary muscle, b) a central area on the left surface of the interventricular septum and c) the posterior "paraseptal" area at about one third of the distance from apex to base. These activated areas increased rapidly in size during the next 5-10 ms, becoming confluent at 15-20 ms. Excitation proceeds from left to right and endocardial excitation in the right ventricular side of the septum (near the insertion of the anterior papillary muscle) does not appear until 5-10 ms after the start on the left side. As activation

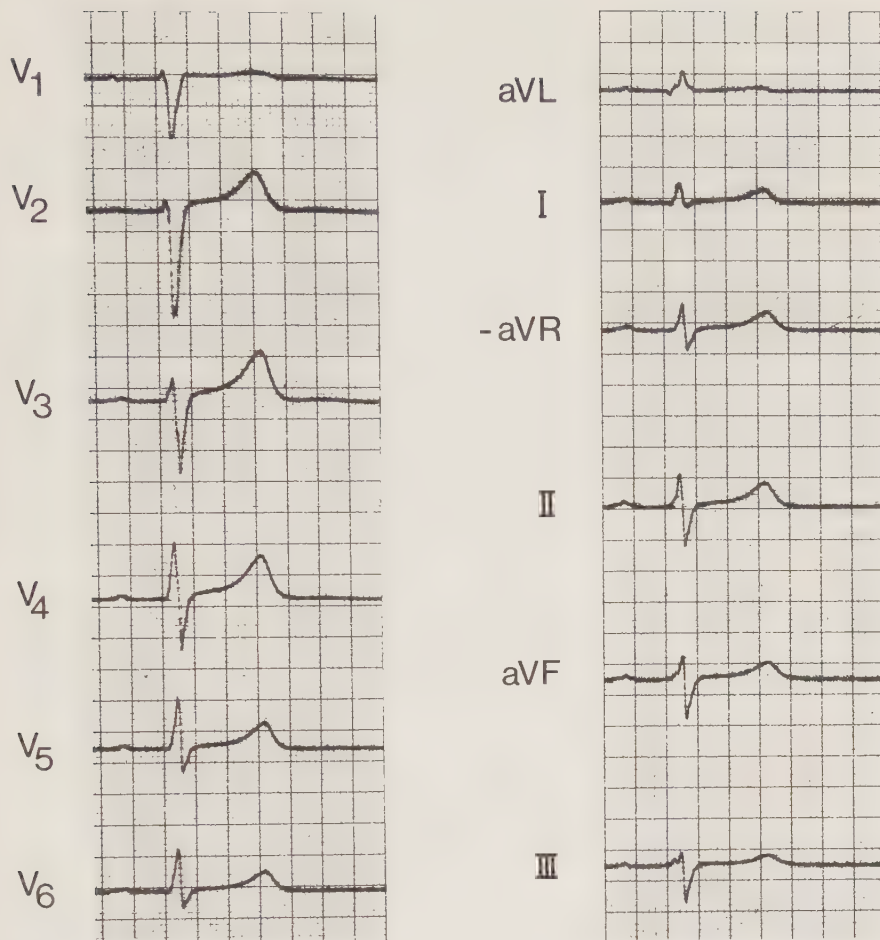


Fig. 7.1. ECG of a man aged 39 years. The mean frontal QRS-axis is less than -30° . The ECG is coded 2.1.1 according to a new ECG-standard, presented in chapter 9. The abnormal electrical position may be due to an unusual anatomical position of this heart, as discussed in the text.

continues, individual variation from the general pattern increases. Therefore, the initial excitation may produce electrical forces, i.e. the Q-wave, that are more closely related to the anatomical structure in which they are produced, i.e. the septum, than the electrical forces produced later. The major electrical forces, which largely determine the mean QRS-axis, are produced 30-50 ms after the start. The Q-axis may therefore be more closely related to the anatomic position of the septum than the mean QRS-axis to the entire left ventricle, at least as long as the septal area is not engaged in a myocardial disease.

In pathological conditions, on the other hand, the septal vector may change direction, depending on the site of damage. The three endocardial areas on the left side of the septum initially excited correspond to the outlets of the three fascicles of the left bundle, i.e. the anterolateral, the posterobasal and the central fascicle (demonstrated by Demoulin et al 1973). Damage to the anterolateral fascicle will change the direction of the septal vector towards the right base of the heart, and damage to the posterobasal fascicle will change it towards the right apex. Q-waves would appear in leads facing the left apex and the left base, respectively. Such changes of direction will be enhanced if the central fascicle is absent, which is the case in about 50 % of normal subjects (Demoulin et al 1973).

Unfortunately, it is not possible to establish with any degree of certainty whether an unusual Q-wave pattern is due to myocardial injury, damage to the fascicles or to an unusual anatomic heart position. It is possible, though, that gross discordance between the Q-axis and the QRS-axis may indicate damage to either the septum or the rest of the left ventricle, while concordance of the two axes suggests an unusual heart position. However, even if direction of the septal vector is subject to variation, its resultant direction will be from left to right in the vast majority of cases. Reversal of the main direction happens only in pre-excitation, CLBBB and in some septal infarctions.

A further argument in favour of the initial vector as a positional marker is provided by the findings of McCaughan et al (1973). In a study of orthogonal ECGs they found that the initial vector (0-20 ms) did not change direction with increasing blood pressure, while the 30 ms vector did.

The Q-axis is a new concept with theoretical advantages as a method to denote the electrical position of the heart, but its value in practical work remains to be elucidated. One practical application has been used in the present study, where normal limits of R and S wave amplitudes have been reported for two separate positional groups differing with respect to the presence or absence of a Q-wave in the lead. In other words, the Q/non-Q-system separates amplitudes in leads facing the left ventricle from leads, which do not.

Much effort has been made in the present study to classify the direction of the Q-axis in positional groups in two planes. One system, for example, implied four positional groups, characterized by the presence and/or the absence of a Q-wave in leads aVL and V₄. Each of these four groups, however, included too many different directions of the Q-axis to be clinically useful. It must be remembered, that some thirty different Q-wave patterns were found in the present study, corresponding to an equal number of different directions of the Q-axis.

The assumption that the Q-axis is related to the anatomical position of the septum implies that the septum can take many different positions. This is in contrast to the findings of Grant (1953), who studied the position of the septum at autopsy. He was not able to demonstrate variation in the septal position. Although, it is unlikely that these observations are relevant evidence for evaluating in vivo conditions, one must be aware of that Q-axis differences in healthy subjects reflect both anatomical and electrical variance between subjects.

The present study indicates that the transitional zone in the precordial leads does not change with age or body build. As for age, this is in line with a study on men (Hiss et al 1960) and on women (Simonson 1961). On the other hand, Simonson (1961) found a slight, but statistically significant shift of the transitional zone to the left with age in men. The influence of body build per se on the transitional zone has not been a matter of large interest in previous studies although some indirect information can be deducted from the relation to the leftward shift of the MFQRS-axis, which is partly an effect of body build. Hiss et al (1960) found no change of transitional zone with a shift of MFQRS-axis to the left, which fits with the present data.

The present study indicates that a transitional zone to the right of V_3 is more common in women than in men. This agrees with the results of Simonson (1961), who also found that extreme CCW-rotation (transitional zone to the right of V_2) as well as extreme CW-rotation (transitional zone from V_5 leftward) were rare in both sexes.

7.2 THE NORMAL QRS-DEFLECTION

The possible usefulness of the Q-wave pattern as an indicator of septal position has been discussed previously. A relationship between the Q-wave pattern (Q-axis) and the mean frontal QRS-axis (MFQRS-axis) was shown in the present study, and this is in line with the findings of Hiss et al (1960). He demonstrated the disappearance of Q-waves in lead III and appearance of Q-waves in lead aVL with a leftward shift of the mean frontal QRS-axis. In other words, Q-waves in lead aVL are more common in horizontal hearts, and Q-waves in lead III are more common in vertical hearts. It also implies that Q-waves are common in leads with large R-wave amplitudes. It has been pointed out that presence of a Q-wave in a lead with a small R-wave should be regarded with suspicion (Sokolow and Friedländer 1949).

The significance of aberrant Q-wave patterns, can not be determined from the present data. Absence of a Q-wave in V_6 appears from VCG-findings to be compatible with septal fibrosis or infarction but may probably also be normal.

The absence of a Q-wave in V_6 in combination with a Q-wave in V_1 appears from VCGs to be a more reliable sign of damage in the septal area. This is also logical, as the two signs are of a nature that can be expected after such a damage. A Q-wave in V_1 alone appears not to be a significant sign of myocardial damage.

The prevalence of Q-wave items according to the Minnesota code was low (1.7 %) in the present study. This is in line with other studies (Blackburn 1960). The Q-wave items, especially 1.1 and 1.2, are among the most reliable items in the Minnesota code as indicators of disease, especially myocardial

infarction, and should consequently be rare in a healthy population like the present one.

In the present study, six subjects were codable according to the Minnesota code Q-wave items. Five of them had code 1.3.4., i.e. a Q-wave of at least 0.03 s in lead III and a Q-wave of at least 1 mm depth in lead aVF. It is important to note that a QIII = 0.03 s is codable according to the Minnesota code and often regarded as abnormal by users of the code. In an autopsy study (Horan et al 1971) subjects with a QIII equal to, or less than 0.03 s had a probability for infarction of 19 %, compared to 75 % for those with a Q-wave larger than 0.03 s. It is most likely that the difference would have been greater if a Q-wave = 0.03 s had been grouped with the abnormal Q-waves, i.e. greater than 0.03 s, as suggested by the Minnesota code. It has been argued, though, that also Q-waves of slightly less duration will be erroneously determined to be 0.03 s if the limit for coding is set to include 0.03 s, instead of more than 0.03 s.

The R-wave

The present study indicates that the R-wave amplitude in men decreases significantly with age especially in inferior, but also in precordial, leads. This is in line with other studies (Simonson 1961 and 1972, Ostrander et al 1965, Harland et al 1967, Blackburn et al 1967). A decrease of the amplitude of the spatial vector calculated from orthogonal leads has been shown by Pipberger (1967) and Simonson (1972). It was also seen in this material. In epidemiological studies, where subjects with disease have not been excluded, the prevalence of Minnesota code 3.1 decreases with age till about 40 years (Ostrander et al 1967, Goldbarg et al 1970, Durakovic et al 1981). After this age the prevalence again rises to the age of about 70 years after which no further rise is observed (Campbell 1974). This latter rise of the code 3.1 prevalence is probably due to a larger proportion of LVH in unscreened populations in older age. The screening of the present material explains why it is not seen in the present study.

It was also found in the present study that R-wave amplitude in men increases with age in leads aVL and I. An increase of the R-wave amplitude in the left limb leads in combination with a decrease in inferior and right precordial leads implies that a change of heart position is an additional factor influencing the R-wave amplitude with age. A shift of the maximal spatial vector with age from an inferior and posterior position to a superior and lateral position was found by Pipberger et al (1967) and Rautaharju et al (1961). Such a shift would explain the age variation of the R-wave amplitudes found in the present study, except for the R-wave in lead V₆, where an increase would be expected with age as a result of the supero-lateral shift. The significant decrease of the R-wave amplitude in V₆, observed in the present study, would have to be explained from other factors such as increased transmission loss with age.

The influence of body build and blood pressure on the mean frontal QRS-axis, as discussed earlier (see page 90), is similar to what we see as age variation. Ischemic heart disease with fibrosis might be a third factor contributing to age variation. It is possible that the age variation is the result of several factors in co-variation. If all factors "work in the same direction", they are likely to result in statistically significant age variation, whereas if they don't, the age variation will be less pronounced. *The less pronounced influence of age in women, found in the*

present study, may be explained by such a mechanism. It was found that obesity did not increase with age in women (which it did in men) and therefore contribution from obesity to age variation of the R-wave amplitude could not be expected in women. Less influence of age in women compared to men has also been indicated in other studies (Simonson 1961 and 1972, Ostrander et al 1965).

However, statistical associations can never prove the causal role of various factors for the age variations observed, and conclusions should be drawn with care.

The present study also shows that R-wave amplitudes are larger in men than in women, especially in younger age. This is well known from other studies of Caucasian subjects (Simonson 1961, Ostrander et al 1965, Jonsson et al 1977, Goldbarg et al 1970, Durakovic et al 1981), as well as in negroes (Walker 1969). The main reason is the smaller hearts of women (Zeek 1942). However, the female breast, as advocated by LaMonte et al (1965) and a generally higher fat content in the female body are also likely to influence the deflection size in the ECG (Simonson 1961). Campbell (1974) found that, in very old people, high amplitude R-waves were more common in women than in men. It is possible that men with LVH die at a younger age than women with LVH.

The mean values and normal limits of the R-wave amplitude found in the present study are of comparable magnitude to those found in other studies of Caucasian subjects (Simonson 1961, Graybiel et al 1944, Harlan et al 1967, Grewin 1948, Blackburn et al 1967). Similar results were also found in studies in non-Caucasian subjects (Burns-Cox et al 1971), although young Chinese men were found to have larger R-waves in the right precordial leads (V_1 - V_3) than the young men in the present study. In the study by Simonson (1961), a few remarkable differences from the present data are to be noted. Upper normal limits (97.5 % percentile) in the present study were generally higher than in the Simonson study, except in lead I in women where Simonson has 16 mm as the upper limit compared to 10 mm in the present study. Especially large R-wave amplitudes in the present study were seen in V_4 - V_5 in young men (36 and 31 mm, respectively) compared to the Simonson study (28 and 24 mm, respectively). In older men, a similar difference was at hand with large amplitude in lead V_4 in the present study (30 mm) compared to the Simonson study (26 mm). Other differences did not exceed 2 mm.

Small differences of normal limits between studies may be explained by differences in sample selection, sample size or reading procedures but larger differences, such as those in lead I in women and in leads V_4 - V_5 in men are more difficult to explain.

7.3 HIGH AMPLITUDE QRS-DEFLECTION IN RELATION TO LEFT VENTRICULAR HYPERTROPHY

The present study indicates that current ECG-criteria for LVH are so frequent in young men that they are clinically useless. This agrees with several other studies (Manning et al 1964, Ostrander 1966, Goldbarg et al 1970, Burns-Cox et al 1971, Östör et al 1981). Furthermore, low sensitivity is a common feature of all the scalar ECG-criteria for left ventricular hypertrophy (Romhilt et al 1969).

Left ventricular hypertrophy is a progressive response of the heart to increased left ventricular work caused by e.g. hypertension or valvular disease. Left ventricular hypertrophy in its early stages is manifested in the ECG as increased deflection amplitudes (Grubschmidt et al 1957). Later on, degenerative changes take place in the myocardium; these are represented in the ECG by ST-segment changes and axis shift to the left (Ackerman et al 1950, Kannel et al 1970, McCaughan et al 1973). With advancing hypertrophy, fibrosis occurs (Ackerman et al 1950, McPhie 1958, Mazzoleni et al 1975). Fibrosis is associated not only with left axis deviation (Grant 1956, Corne et al 1965) and ST-segment changes (Weinberg et al 1950), but also with diminished QRS-amplitudes (Weinberg et al 1950). It is therefore likely that some advanced hypertrophies are to be found in ECGs with lower amplitudes.

The reduction of the amplitudes with fibrosis has not been taken in account in any of the current criteria for LVH. This may be one reason for the low sensitivity found in autopsy studies, i.e. as low as 40-54 %, even with the system of Romhilt and Estes (Romhilt 1969, Geva 1979), which is considered the best one. The specificity of this system in men is as high as 96-100 % (Romhilt 1969, Geva 1979).

In order to improve the sensitivity, a new code for left ventricular hypertrophy is proposed in the present study. (Table 5.III). It is based on R-wave amplitudes in the four leads aVL, I, V₆ and V₅ and the S-wave amplitude in V₁. Consideration has been taken not only to sex, age and heart position, but also to the presence or absence of factors likely to be associated with fibrosis, i.e. ST-segment changes and left axis deviation. Amplitude limits have been adjusted to fit into an arithmetic series in order to be easily remembered. The code was developed from the upper normal limits in the present study, and was then tested in an unrelated sample of men and women with high and low casual blood pressure. The new code appears to have better sensitivity than the Romhilt and Estes system, but nevertheless provides better specificity than the Minnesota code. Although final measurements of sensitivity and specificity can be obtained only in a larger study where preferably also left ventricular hypertrophy can be measured, it appears that the new code may be more useful than other coding systems in the evaluation of hypertensive patients.

Let us first discuss the importance of early diagnosis of left ventricular hypertrophy (LVH) and how to validate criteria for this diagnosis. LVH in this study is an electrocardiographic diagnosis. LVH by ECG in the Framingham study (Kannel et al 1970) was associated with a grave prognosis as regards evolution of ischemic heart disease. Unfortunately, the amplitude criteria and ST-segment changes were not very well defined (Kannel et al 1969), but presumably very pronounced as indicated in a later study (Kannel et al 1970). Minor deviations from normal are not usually considered in large population studies, such as the Framingham study. In the Framingham study, LVH was denoted as either possible or probable LVH, depending on the absence or the presence of a typical left ventricular strain pattern, respectively. Minor ST-changes were not accepted as a typical left ventricular strain pattern and LVH with such minor ST-changes were consequently designed as possible rather than probable LVH. It is therefore likely that the diagnosis of LVH, possible as well as probable, was made at a time when the hypertrophy was pronounced and had reached an irreversible stage, which would explain the poor outlook for the subjects with LVH by ECG.

For early diagnosis, the clinician needs a finer instrument than the rather coarse ECG-criteria, commonly used in large epidemiological studies. Furthermore, he does not only need criteria with finer gradation of changes, but criteria, which are adapted to sex and age variations. With such criteria the diagnosis of LVH could be made at an earlier stage, when it is reversible. Reversibility of LVH after treatment has been shown for the ECG-signs (Ibrahim et al 1977, Tarazi 1983, Kannel et al 1970 and 1983, Freis 1983) as well as for the echocardiographic signs of LVH (Drayer et al 1983, Wollam et al 1983). A beneficial effect of antiadrenergic treatment has also been shown in animal studies (Subha 1983, Pegram et al 1983).

As mentioned, early diagnosis of LVH is important. Cut off points in the ECG for such a diagnosis can be derived from either studies of hypertrophied hearts or from studies of normal subjects, as has been done in the present study. Validation of the cut off points could be done by measurement of the left ventricular weight at autopsy, or by evaluation of the left ventricular mass by echocardiography or other methods. Validation could also be done by comparing the cut off points with other ECG-criteria, which have known sensitivity and specificity. If a new criterion appears to be better compared to an old one, this should be confirmed by either an autopsy or an echocardiographic study. The validity of the new code A, introduced in the present study, has not been confirmed in this way. The value of code A at this stage is not to diagnose anatomic LVH, but rather to classify ECGs into either of two groups, namely those with normal amplitudes and those with abnormally high amplitudes. Those with abnormally high amplitudes have a probability of less than 2.5 % to be normal.

The new code differs in two respects from previously proposed criteria. Firstly, the new code is adapted to age and sex variations. This is essential for the accuracy of any hypertrophy criteria. Only age has been considered before, in some programs for computer-assisted interpretation (MacFarlane 1971). Secondly, the new code accepts lower amplitude limits as indicative for left ventricular hypertrophy in the presence of ST-segment changes and left axis deviation, which are known to be associated with low voltage. This approach to the diagnosis of LVH appears not to have been advocated previously.

Finally, it is of interest to compare the performance of this new scalar ECG-code with an orthogonal ECG-code. Orthogonal codes are regarded as more sensitive than scalar codes (MacFarlane et al 1981). Wikstrand et al (1976) studied 50 year old men in Gothenburg. He applied the orthogonal criteria of McCaughan et al (1973) in two groups of subjects with differing blood pressure. The group with high blood pressure had a higher mean blood pressure (200/120 mm Hg) than the "hypertensive" women used in the present study (mean BP = 175/100 mm Hg). In spite of this, Wikstrand et al scored only 33 % of their ECGs "positive" using orthogonal leads, while in the present study, 40 % of the hypertensive women were "positive" using the new scalar code. The group with lower blood pressure in the Wikstrand study had a mean blood pressure (140/90 mm Hg) similar to the "hypertensive" men of the present study. The orthogonal code identified only 3 % of their ECGs, while the scalar code identified 10 % of corresponding ECGs in the present study. Although it is difficult to compare studies with different methodology and material, *it seems likely that the new scalar code presented here is more sensitive than the orthogonal code used by Wikstrand et al (1976).* Orthogonal codes, however, may be improved by giving consideration to the variation with sex and age.

There are two additional features worth commenting upon in context with LVH namely the left atrial involvement and the ventricular activation time (VAT).

Left atrial involvement had been demonstrated in subjects with valvular disease in the left heart (Morris et al 1964). The left atrial involvement, expressed as the P-terminal force in lead V_1 (that is terminal $P_{V_1} \geq 0.04$ s and ≤ -0.1 mV) correctly separated patients without valvular disease from those with left valvular disease in 92 % of 200 subjects. The Morris P-wave criterion, as defined above, has been widely applied in clinical practice, as an indirect sign of LVH. Furthermore, it may be assumed that it does reflect a later rather than an early LVH.

The ventricular activation time (VAT) is the time from onset to the maximum of the QRS-deflection. Prolongation of this time to 0.05 s or more in left ventricular leads have been taken as a sign of LVH (i.a. Romhilt and Estes 1968). It is likely that also the VAT-prolongation represents a more advanced stage of LVH, that is when influence on the right ventricle has taken place. In an autopsy study by Carter and Estes (1964) it was found that VAT correlated significantly with total heart weight and right ventricular wall thickness in hypertrophied hearts but not with left ventricular thickness. It is likely that electrical forces from a hypertrophied right ventricle will counteract the electrical forces from the left ventricle for a longer period of time, thereby decreasing the resultant electrical forces during this period. This could theoretically result in a prolonged VAT, as unopposed forces from the left ventricle will appear later than usual.

In chapter 9 an ECG-standard for clinical use will be proposed and the Morris P-wave criterion and VAT will be included in the hypertrophy item together with the amplitude criteria of code A.

7.4 THE NORMAL REPOLARISATION DEFLECTION

The ST-segment is difficult to evaluate without the use of a classification system. The ST-segment is not a single amplitude measurement, but instead a great number of amplitude measurements on a time scale. Classification of the ST-segment implies that the form and direction of the ST-segment can be described from two amplitude measurements, namely beginning (ST-junction) and end (beginning of the T-wave) of the ST-segment and the relationship between them. Here the end of the ST-segment is particularly difficult to define. Nevertheless an ST-segment can be classified as being ascending, horizontal or downward sloping with different degrees of ST-junction depression.

Attempts to analyze the cause of ST-changes have involved a more and more detailed classification. The original Minnesota code had three categories. Additions have mainly come from Scandinavian countries (Scandinavian Committee 1967) culminating in the detailed system proposed by Punsar et al in 1968 (Fig. 2.5). Driving forces behind this development have been the use of CR-chest leads that was previously common in Scandinavia, and the frequent use of exercise testing.

The "Punsar code" has three main categories, ischemic (horizontal or downward sloping), slowly ascending and rapidly ascending ST-depressions. During a 5 years follow-up study the ischemic codes (I1-I4) were associated with a significantly higher rate of cardiac deaths than all the other codes (Punsar et al 1968). Code S4 was also associated with a higher cardiac mortality ($\chi^2 = 8.75$, $p < 0.01$) and the difference was still significant if I5 was included ($\chi^2 = 5.95$, $p < 0.05$). There was great inter-observer variation for ST-segments with a junction depression of less than 0.5 mm (I5 and S4). Codes S1-S3 were associated with more "coronary" ECGs after 5 years ($\chi^2 = 31.0$, $p < 0.001$) but no cardiac deaths were registered.

The predictive value of codes R1-3 in the resting ECG is unknown because the group in the Punsar study was too small ($n=9$). The codes R1-3 after exercise had the same favourable prognosis as the non-codable ECGs.

In the present study, a surprisingly high prevalence (16 %) of ECGs with horizontal or downward sloping ST-segments (HoDST) was found if all the "Punsar leads" were used (i.e. aVL, I, II, aVF, V2-V6). The reason was, that such ST-segments were common in aVL and aVF. If these two leads were omitted from the classification system the prevalence of HoDST was reduced to 5.3 %. The high prevalence of HoDST in leads aVL and aVF suggest a non-pathological origin. If the benign nature of ST-segment changes in these leads could be verified in follow-up studies, it seems reasonable to omit them as "qualifying leads" in the Punsar classification system.

The present study shows that slowly ascending ST-segments are significantly more common in women than in men, whereas no differences were found as regards the ischemic ST-segments. It has not previously been found, that the sex difference of the ST-segment, is confined to the slowly ascending ST-segment.

The higher prevalence of slowly ascending ST-segments in women may be a consequence of the generally lower amplitudes in women compared to men. With lower R- and T-wave amplitudes it is natural that also ST-junction amplitude and ST-segment inclination is lower in women than in men. This is in line with other studies on Caucasian subjects (Kossman 1953,

Walker et al 1969) and on Negroes (Walker et al 1969, Gottschalk et al 1956, Seriki et al 1966, Grusin 1954).

The smaller ECG-amplitudes in women can be related to a smaller heart muscle, reflected in a smaller stroke volume (Astrand 1970) and to less good transmission properties through the body caused by a higher fat content (Simonson 1961) and the breasts (LaMonte 1965).

It is important to note that no significant sex differences were found as regards the ischemic ST-segment changes in the present study and this is in contrast to other studies, in which the Minnesota code (Rose and Blackburn 1968) or the modified Minnesota code (Scandinavian committee 1967) have been used (Ostrander et al 1965, Goldbarg et al 1970, Campbell et al 1974, Jonsson et al 1977). In these studies a female dominance was observed also for ischemic ST-segments. The reason for this discrepancy may be different sample sizes. However, it is hardly possible to compare these studies with the present one, as the prevalence of slowly ascending ST-segments cannot be evaluated from either the original or the modified Minnesota code.

As mentioned, Punsar et al (1968) found that ischemic and slowly ascending ST-segment in men were associated with higher cardiac morbidity and mortality. It appears that the pathological significance of similar ECG-changes are different in women.

Women have been found to have a high rate of ST-segment changes in exercise ECGs (Cumming et al 1973) but the well-known lower incidence of myocardial infarction in young and middle-aged women, as compared to men, suggests strongly that the ST-changes are not predictors of coronary heart disease in the same way as in men.

In the present study, it was found that the prevalence of ischemic and slowly ascending ST-segments increased with age only in men. Age trends in the ECG have been taken as signs of ischemic heart disease (IHD) in a population (Simonson 1972). The finding that ST-segment depression increased with age may be explained by the higher incidence of IHD in men compared to women. In studies of total populations, however, age trends have been observed also in women (Ostrander et al 1965, Goldbarg et al 1970).

There is a strong relationship between ST-segment depression and heart disease. Blackburn et al (1960) compared subjects without and with infarction. In a population with known infarction the prevalence of codes 4.1-3 was 26 %. In population studies in the United States and Yugoslavia on apparently healthy subjects the prevalence ranged from 1.6-2.9 %. In railway employees, carefully screened for disease, the prevalence was as low as 0.4 %. The present study is also very carefully screened for disease but the prevalence of Minnesota code 4.3 (i.e. Punsar code I4) was 2.5 % among the men. Minnesota code 4.1-2 was not present at all.

Short (1972) studied minor ST/T-depressions in the resting ECG. The ST-item studied corresponded to the Punsar code I4-5. In addition, the Punsar S4 was probably included as judged from the illustrations in the original paper. In 200 apparently healthy men and women (students) under the age of 30, these items were not found at all. In a population of 225 patients with a prevalence of heart disease of 92 %, the codes mentioned were present in 2/3 of the population. The striking difference between the two populations was taken to support the conclusion that presence of the defined items in

a resting ECG is abnormal. In the present study the prevalence for items I4-5 was 3 % in men and 9.8 % in women under the age of 30 in comparable leads.

Oberman et al (1972) studied the natural history of coronary artery disease in a group of patients with angiographically proven coronary artery disease. They found that the presence of Minnesota code 4.1-3 doubled the mortality during a follow-up period up to 4 years, in those patients with two or three vessel disease.

The T-wave amplitude was found to be larger in men than in women in the present study. This agreed with the findings of Simonson (1961), who also found a significant decrease of the T-wave amplitude with age especially in men. The age trend in men has been demonstrated in the orthogonal ECG by Pipberger et al (1967). They found a 6 % decrease of the amplitude with each decade of life. *In the present study, variation of the T-wave amplitude with age and heart position was significant in most leads,* although the differences were very small when normal limits were evaluated.

The normal limits of the T-wave amplitude for men in the present study were found to conform with those of Leatham (1950) and Burns-Cox et al (1971), and also conform reasonably well with those found by Simonson (1961). The upper limits were as a whole slightly larger in the present study compared to the study by Simonson. The differences were hardly of practical importance. Also the lower limits conforms well, which is somewhat surprising as the two studies use different reference lines, namely the TP-segment in the study by Simonson and the PQ-segment in the present study.

In the review by Kossman (1953) of older normal series the amplitudes of the upper limits are generally much larger than those found in the present study and the lower limits are either flatness or inversion. Inversion was found not only in the leads aVL, III and V₁ as in the present study, but also in leads I, aVF and V₂-V₄. The explanation for these differences is probably to be sought in differences in reading procedures and the quality of the ECGs forty years ago compared to today's standardized procedures and computer-averaged ECGs. Another explanation may be differences in sample composition with inclusion of negroes in the older materials. Higher T-wave amplitudes have been found in negroes (Goldman 1953, Walker et al 1969, Pipberger et al 1967) and also a persistent juvenile pattern with inverted precordial T-waves (Littman 1946, Grusin 1954, Walker et al 1969). Thus, ethnic factors may explain the differences. However, in another study (Burns-Cox et al 1971) of young Chinese and Malay men, ethnic differences were not seen and the upper as well as the lower limits conformed well with the ones in the present study.

The T-wave is affected in many types of cardiovascular diseases (e.g. Blackburn et al 1960, McPhie 1958, Short 1972, Astrand et al 1965, Sokolow and Lyon 1949, Rautaharju 1961). The theoretical background of the T-wave has been reviewed by Noble and Cohen (1978). Even pronounced T-wave changes may occur without heart disease. Of particular interest is the effect of sympathetic stimulation (e.g. Nordenfelt 1941, Johansson 1957, Taggart et al 1979, Atterhög et al 1982, Abildskov et al 1970) and hyperventilation (Simonson 1961, Kemp et al 1968, Lary et al 1976, Joy et al 1981). Such effects may be of great importance clinically but would probably be of small influence under the present measuring conditions. "Unspecific" T-wave changes were common in young men and the prevalence decreased to the

age of 35 years (Hiss et al 1962), after which age the prevalence again increased. The latter increase with age probably reflects the increasing prevalence of IHD. This view is favoured in other studies as well (Blackburn et al 1967 and 1970, Goldbarg et al 1970, Ostrander et al 1965).

The prevalence of T-wave items according to the Minnesota code 5.1-4 was low, but appeared larger in men compared to women in the present study. In a study by Jonsson et al (1977) of randomly selected men and women aged 18-65 years the prevalence of the modified Minnesota code 5.1-4 was 2.5 % in men and 1.7 % in women, which is in line with the findings in the present study, where comparable prevalence figures were 4.3 % and 2.1 % respectively. The agreement between those two studies is surprising, as different lead systems (CR and V leads, respectively) and consequently different coding systems have been used. In the study by Jonsson the Scandinavian adaptation to CR-leads (1967) was used while in the present study the original revised Minnesota code (1968) was used. Code 5.4 on low amplitude T-waves differs between the two systems. In the original Minnesota code the amplitude is expressed in relation to the preceding R-wave while in the Scandinavian modification it is expressed in absolute standards as < 2 mm in leads I, II and CR₂₋₇. If this code was applied to leads I and II in the present study, the prevalence of low amplitude T-waves in lead I should be 41 % and in lead II 17 %. This finding can hardly be explained by the small difference in base line (T-P in the Scandinavian modification and PQ in the present study). The code 5.4 of the Scandinavian modification appears not to be relevant as concerns leads I and II.

In children the T-waves over the right precordium are often negative. It is known that this juvenile pattern may persist in adults, particularly in Negroes (Littman 1946, Grusin 1954, Gottschalk et al 1956, Walker et al 1969) and women (Simonson 1961, Gottschalk et al 1956, Ostrander et al 1965).

In the present study negative T-waves over the right precordium were confined to lead V₁ and were far more common among women than in men, and the T-wave inversion rarely exceeded 2 mm. Negative T-wave in both V₁ and V₂ was found only in one woman.

The present study reports normal values for the T/R-amplitude ratio. It has been suggested that decrease of the T/R-ratio is a harbinger of ST-segment depression (Sokolow and Lyon 1949). It has also been shown that elevation of the arterial blood pressure results in decrease of the T/R-ratio (Rautaharju et al 1961, Ostrander 1966). On the other hand, McPhie (1958) found that decrease of the T/R-ratio expressed ischemic heart disease rather than left ventricular hypertrophy.

8 POSSIBLE SILENT HEART DISEASE IN THE PRESENT MATERIAL

Throughout the present study it has been natural to look at the VCG when an unusual feature was found in the ECG of a subject. It was soon decided, in order to avoid bias, that all VCGs should be analysed independently. However, as the VCG is of the same basic nature as the ECG, it was also decided that VCG could not be used as a criterion for exclusion of ECGs from this study, but only to shed some additional light on ECGs with unusual features, that is ECGs outside or defining the normal limits.

The present accepted sample (n=357) consists of subjects, who were presumably healthy, as they did not have any signs or symptoms of heart disease. However, atherosclerosis of the coronary arteries starts early (Lober 1953) and significant stenosis (> 50 %) has been found in 12-25 % of young physically fit men (Enos et al 1953, Mason 1963). Total occlusion of one or more vessels were found in 3 % of young soldiers, killed in action in Korea (Enos et al 1953). Nothing is known about the ECGs of these subjects, but it can be assumed that the occlusions had taken place without symptoms, or else the subjects would not have been selected for their mission.

It is therefore likely, that subjects with silent heart disease have been included in the present, presumably healthy sample. The prevalence of possible silent heart disease has been evaluated by analysis of the VCGs and well known criteria of heart disease (Arvill et al 1972, Starr et al 1974, Benchimol 1979, Cowdery et al 1980) have been used.

The VCGs were analysed by one experienced observer without knowledge of the appearance of the scalar ECG, but with knowledge of age and sex of the subject. The VCGs were labelled definitely abnormal, probably abnormal, probably normal or definitely normal VCG. The evaluation of the VCGs was lenient, as the expected prevalence of disease was low.

Definitely abnormal QRS-loops on VCG (DA-VCG) were found in 5 of the 163 men (3.1 %). ECG, VCG and diagnosis of these subjects are reported in Figs. 8.2, 8.3, 8.6, 8.8 and 8.9. DA-VCGs were not found among the women.

Probably abnormal QRS-loops on VCG (PA-VCG) were found in eight men (4.9 %) and in five women (2.6 %). Thus, altogether 18 subjects (13 men and 5 women) had VCGs, which indicated possible heart disease.

It would be interesting to see, to what extent these subjects have influenced the setting of normal limits of items in the scalar ECG in the present study. Therefore, 95 % confidence interval of all items were calculated for the 150 men and 189 women with normal or probably normal VCGs. It was found that this interval as concerns Q-wave durations, Q-wave amplitudes, Q/R-ratios, R-wave amplitudes, S-wave amplitudes, ST-junction amplitudes, T-wave amplitudes and T/R-ratios showed no important differences from the normal values obtained when the whole accepted sample (163 men and 194 women) was analysed. On the other hand, a prominent influence was found on the left limit of the mean frontal QRS-axis (MFQRS-axis) in men and on the right limit of the precordial transitional zone (TZ).

The normal limit to the left of the MFQRS-axis was calculated to -15° for men, aged 35 years or older (Table 3.1). If subjects with DA-VCGs or PA-VCGs were excluded, the normal limit to the left would be 0° . In Fig. 3.2

(page 45) subjects with DA-VCGs (subjects 4-5) and PA-VCGs (subjects 3, 6 and 7) are noted. The ECGs and VCGs of these subjects are shown in Figs. 8.1-8.5.

The normal limit to the right of the precordial TZ was calculated to be between leads V_1 and V_2 . If one man with DA-VCG (Fig. 8.6) and one man with PA-VCG (Fig. 8.7) and one woman with PA-VCG (Fig. 8.10) were excluded the normal limit to the right would be a precordial TZ in lead V_2 .

Thus it has been found that three of the five DA-VCGs and five of the eleven PA-VCGs have had a major influence on the normal limit of the heart position in the present study, but no other normal limits have been significantly influenced by these subjects.

In the next chapter, a new ECG standard for clinical use will be proposed. That standard is based on the normal limits of the present study as found in chapters 4 and 6 but consideration has been given to the VCG-findings in context with the heart position items.

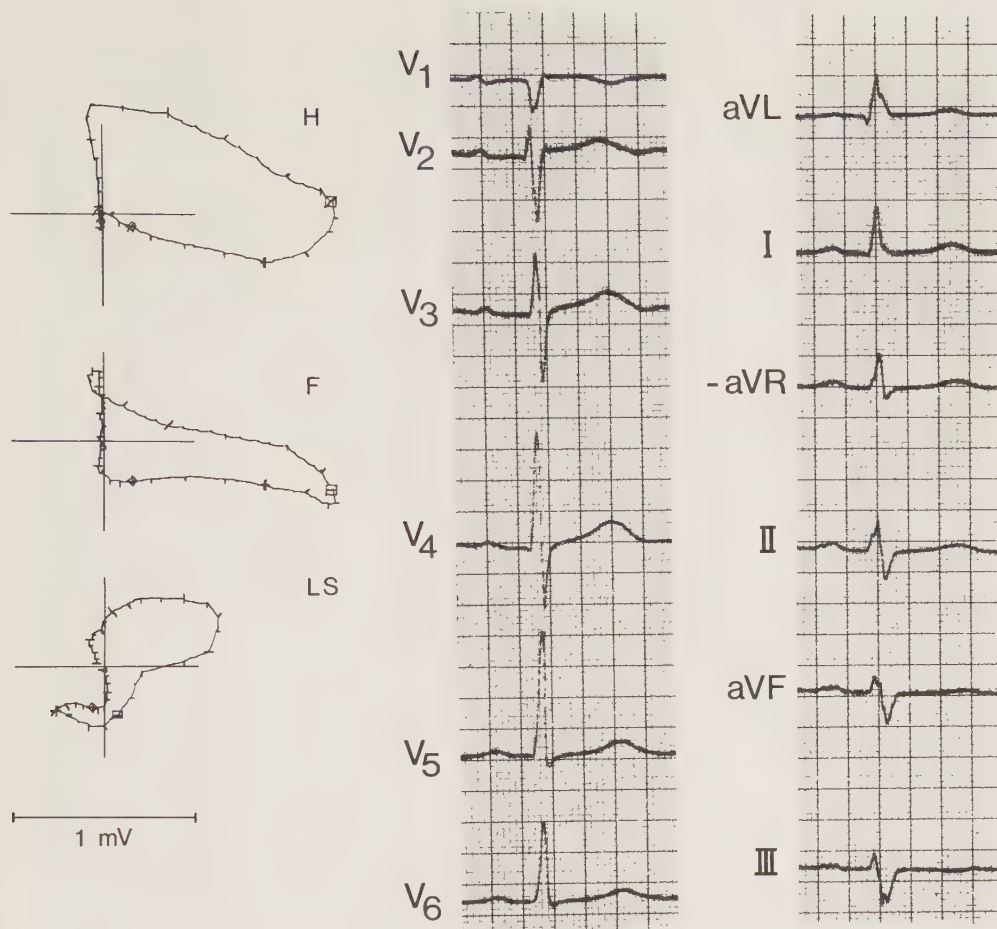


Fig. 8.1. Probably abnormal QRS-loop on the VCG of a man aged 39 years (= subject No. 3 in Fig. 3.2). Initial CW-rotation in the horizontal (H) and left sagittal (LS) planes and an anterior bite in the loop 0-25 ms indicate a small anteroseptal myocardial infarction.

F = frontal plane.

◇ = 20 ms vector, + = 30 ms vector, □ = 40 ms vector.

Other time marks correspond to 2.5 ms.

The scalar ECG demonstrates LAD of -30° and an unusual Q-wave pattern with a small Q-wave in lead V1 but no Q-wave in V6. This ECG is coded 1.4 and 2.1 according to the new classification system presented in chapter 9.

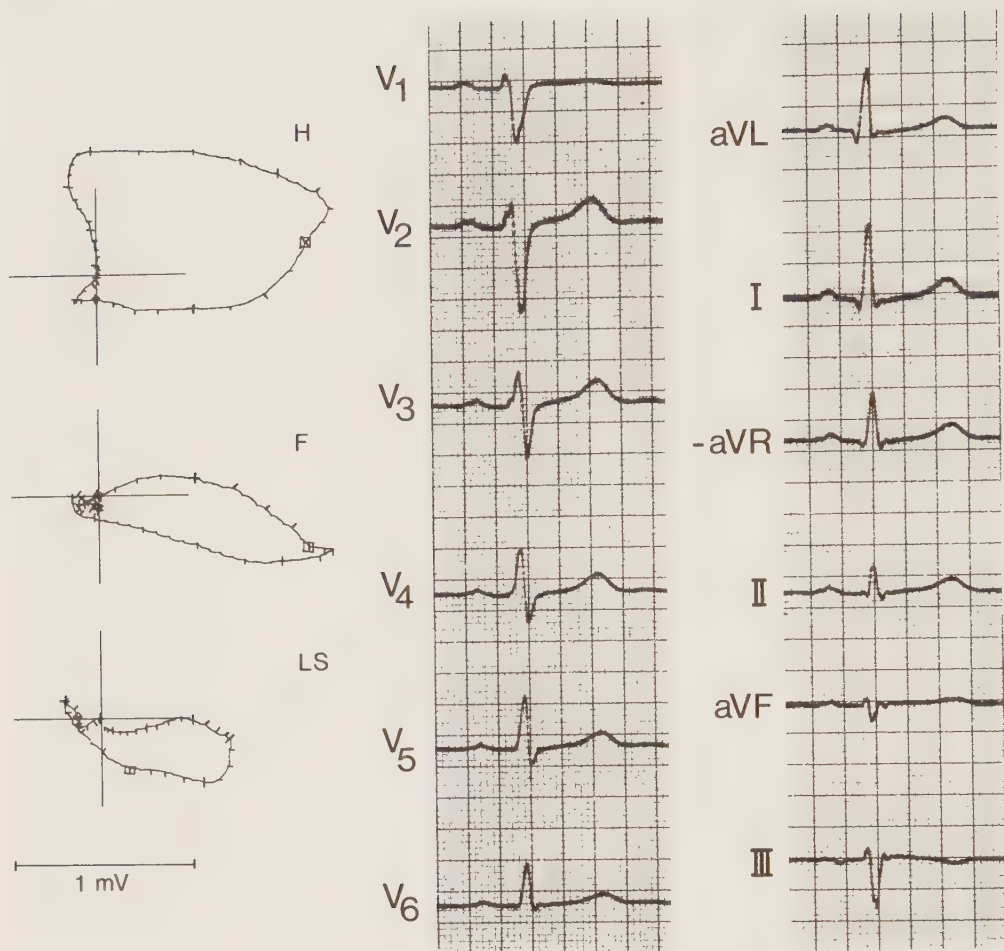


Fig. 8.2. Definitely abnormal QRS-loop in the VCG of a man aged 43 years (= subject No. 4 in Fig. 3.2). Superiorly directed vectors at 20-32.5 ms indicate inferior myocardial infarction.

H = horizontal plane, F = frontal plane, LS = left sagittal plane.

◇ = 20 ms vector, + = 30 ms vector, □ = 40 ms vector.

The scalar ECG demonstrates LAD of -15° . ECG code 2.1 (chapter 9).

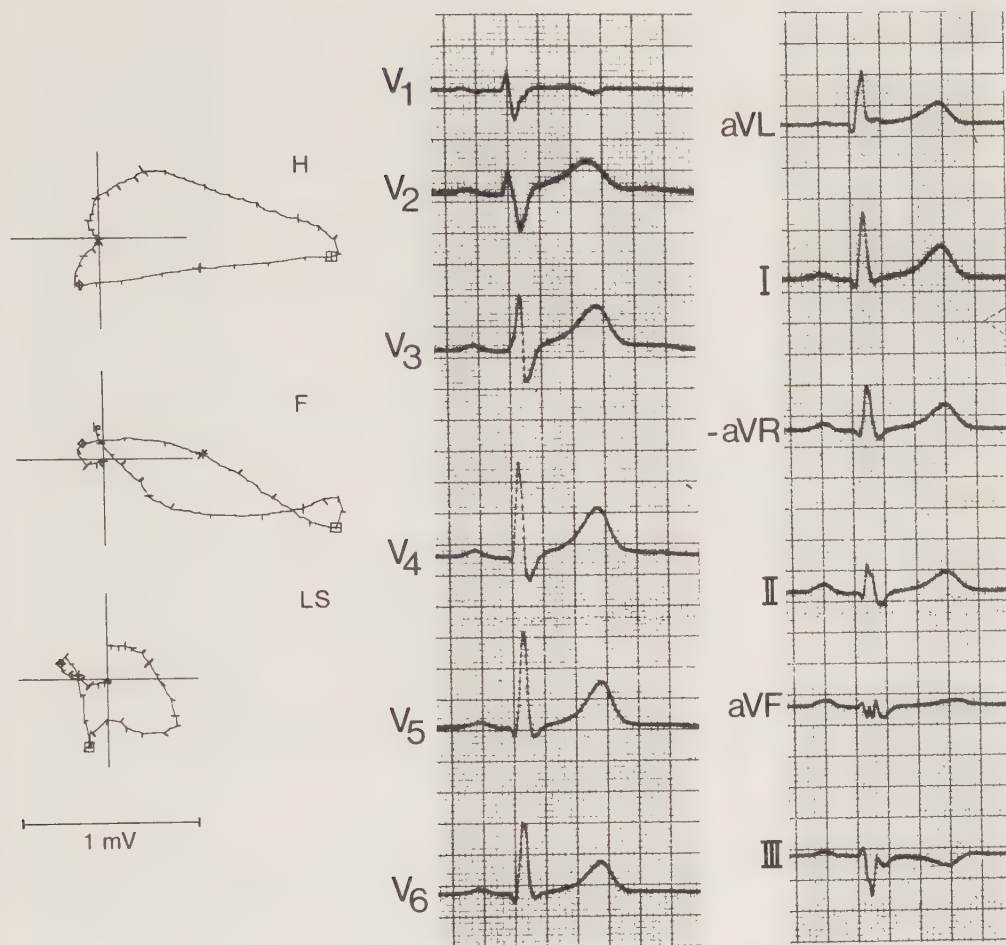


Fig. 8.3. Definitely abnormal QRS-loop on the VCG of a man aged 47 years (= subject No. 5 in Fig. 3.2). Initial CW-rotation in the left sagittal plane (LS) 0-40 ms with a figure-of-eight and superiorly directed vectors during 22.5-30 ms indicate inferior myocardial infarction.

H = horizontal plane, F = frontal plane.

◇ = 20 ms vector, + = 30 ms vector, □ = 40 ms vector.

The scalar ECG demonstrates LAD of -15° . The T-wave in lead III is -1.8 mm, which exceeds the normal limit (-1.0 mm). ECG code 2.1 (chapter 9).

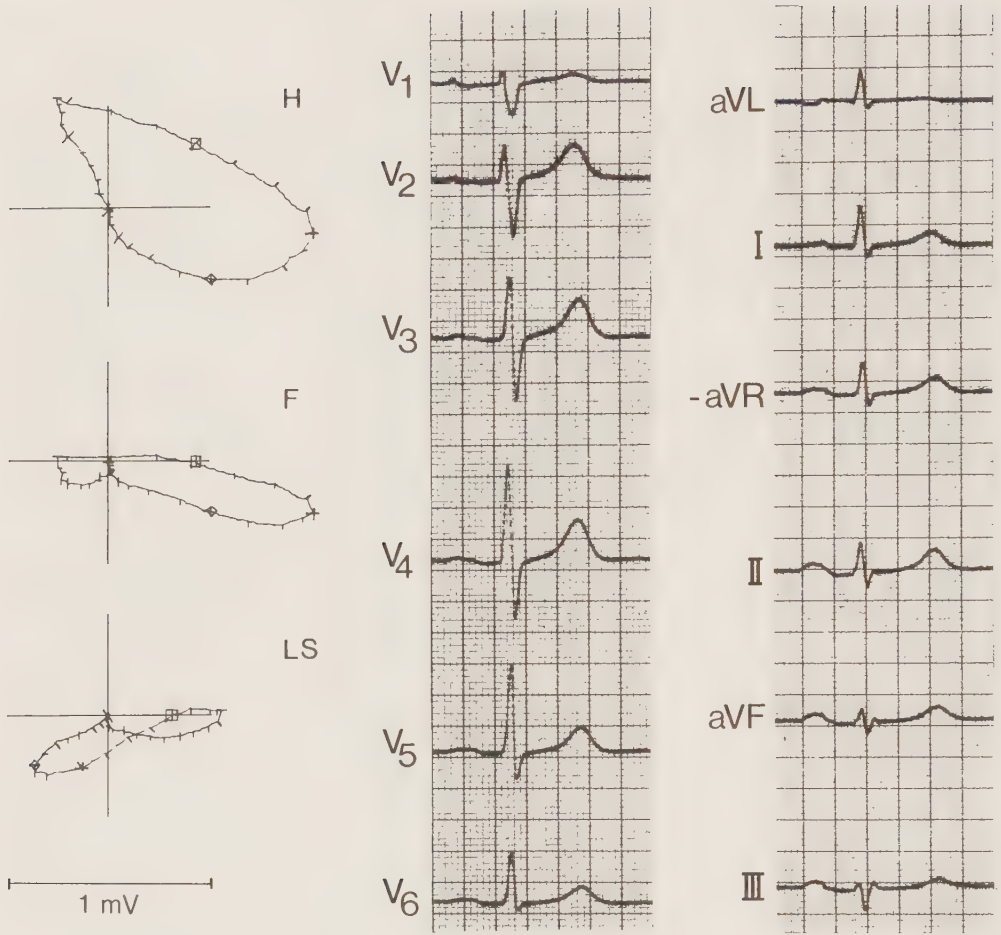


Fig. 8.4. Probably abnormal QRS-loop on VCG of a man aged 55 years (subject No. 6 in Fig. 3.2). Relatively large defect inferiorly with a figure-of-eight in the left sagittal plane (LS) indicate inferior myocardial infarction.

H = horizontal plane, F = frontal plane.

◇ = 20 ms vector, + = 30 ms vector, □ = 40 ms vector.

The scalar ECG demonstrates a MFQRS-axis of 0° .

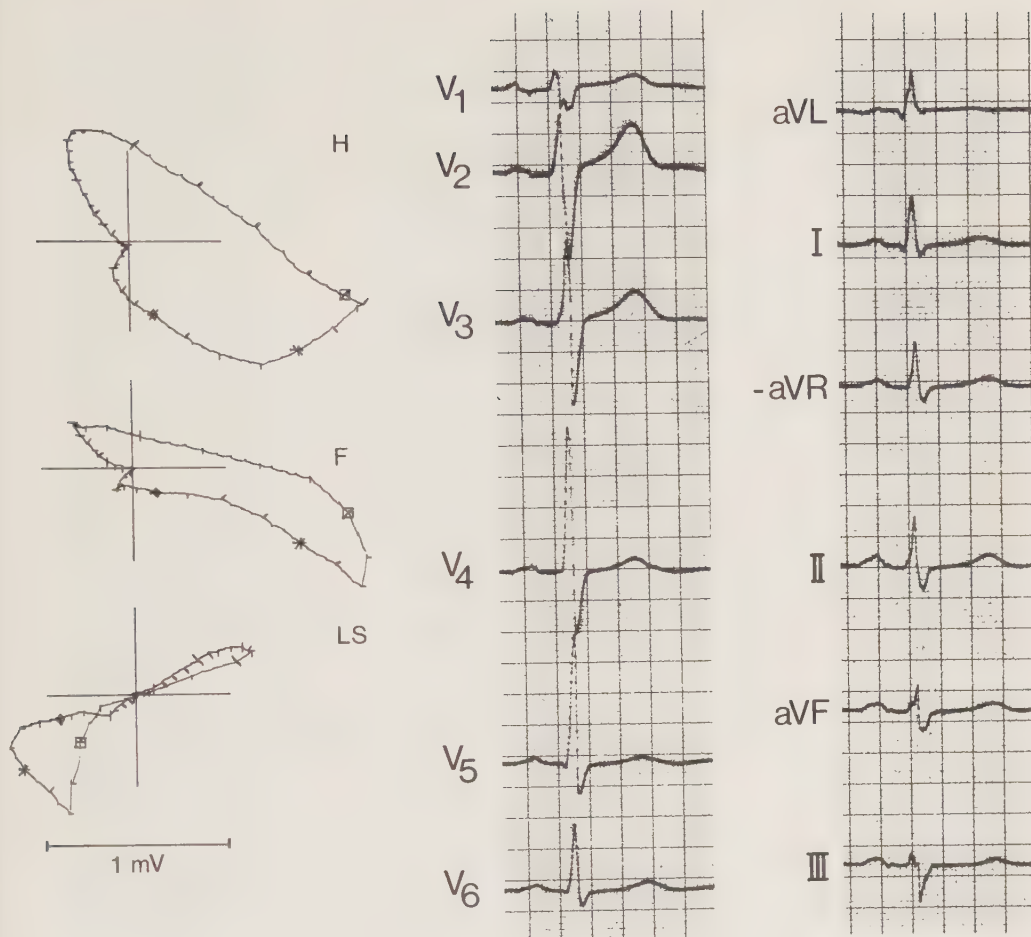


Fig. 8.5. Probably abnormal QRS-loop on VCG of a man aged 57 years (= subject No. 7 in Fig. 3.2). Large posterior defect with a figure-of-eight in the left sagittal plane (LS) and anterior dominance of the loop area in the horizontal plane (H) indicating posterior infarction. F = frontal plane.

◇ = 20 ms vector, + = 30 ms vector, □ = 40 ms vector.

The scalar ECG demonstrates a MFQRS-axis of 0° and also note the unusual appearance of QRS in lead V_1 with an R/S-amplitude ratio close to one. The notched, shallow but wide S-wave reflects the loss of posterior forces in this ECG.

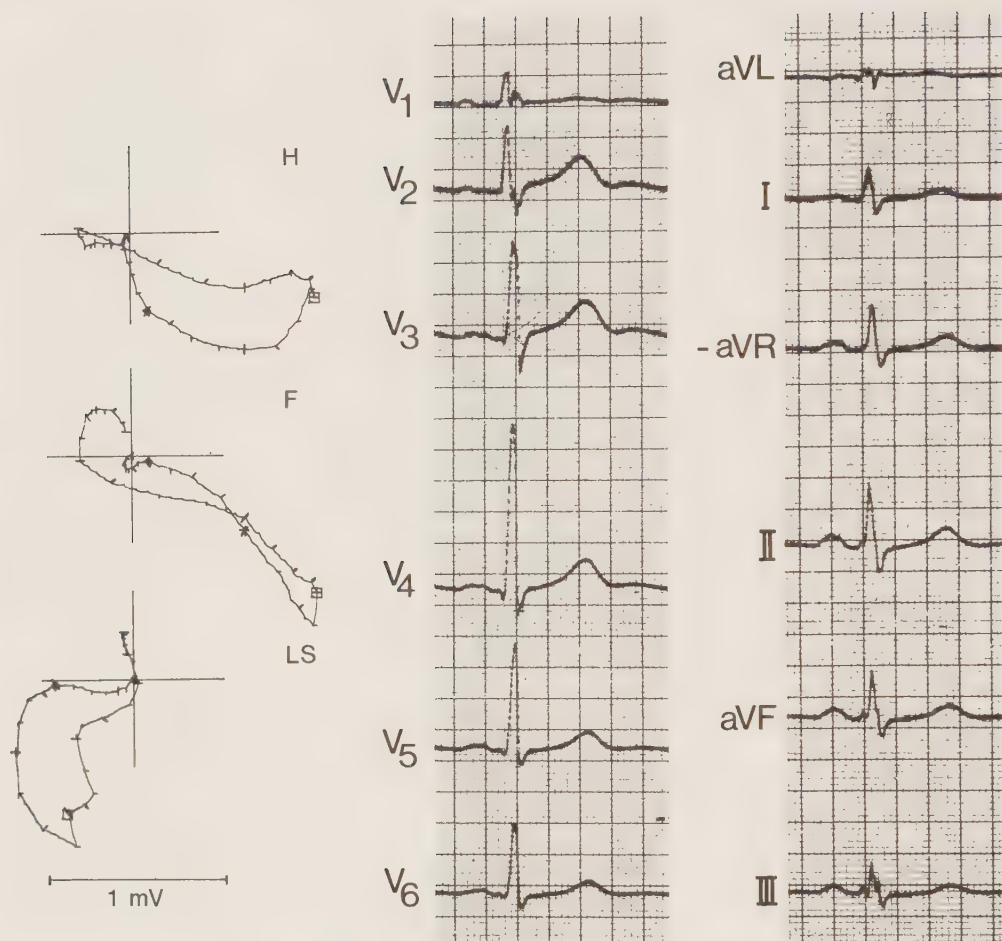


Fig. 8.6. Definitely abnormal QRS-loop on VCG of a man aged 33 years. Large posterior defect with almost no posteriorly directed vectors and a figure-of-eight in the horizontal plane (H), indicating posterior infarction.

F = frontal plane, LS = left sagittal plane.

◇ = 20 ms vector, + = 30 ms vector, □ = 40 ms vector.

The scalar ECG demonstrates a precordial transitional zone to the right of lead V₁. The S-wave amplitude in lead V₁ = 0 mm and in lead V₂ = 3.2, which both are below the lower normal limits of 4 mm in these two leads. The ECG is coded 2.4 (chapter 9).

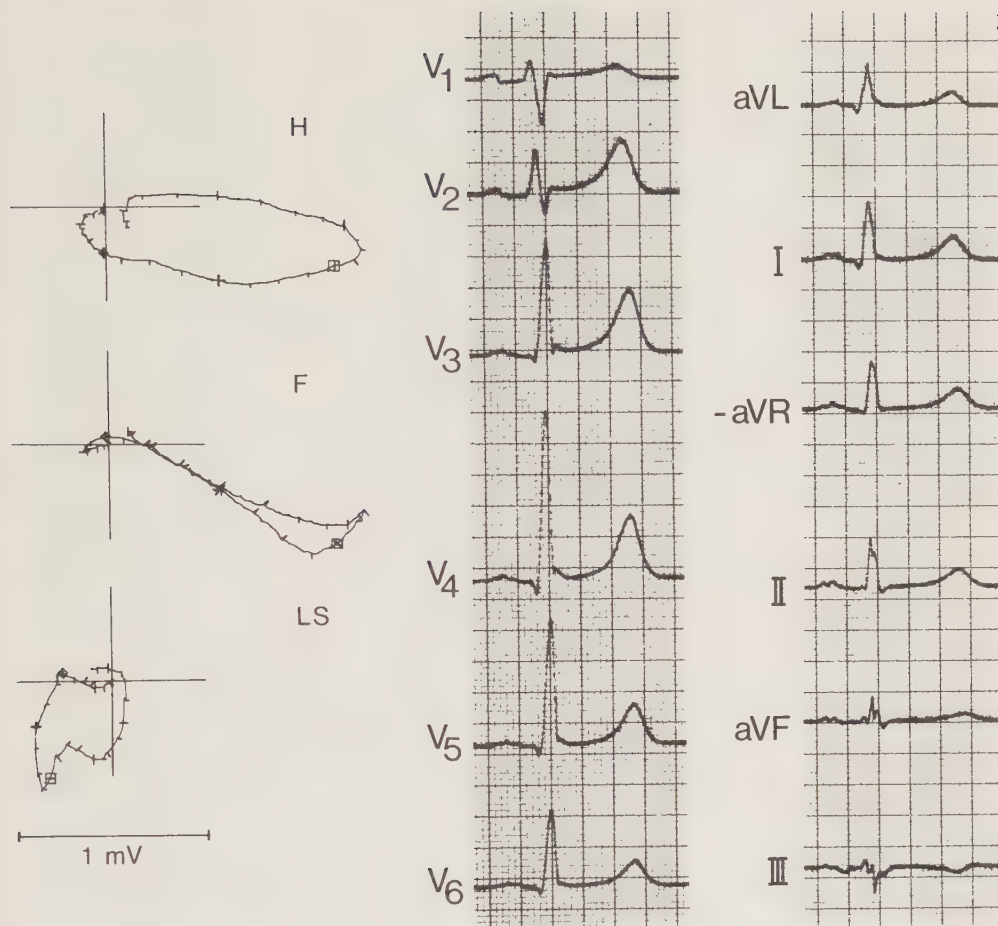


Fig. 8.7. Probably abnormal QRS-loop on VCG of a man aged 52 years. Moderate bites in the QRS-loop in the left sagittal plane (LS) and frontal (F) plane and an anterior dominance in the horizontal plane (H), with respect to time as well as loop-area. The vectors are directed anteriorly during the initial 60 ms. The VCG indicates possible posterior infarction. $\diamond$ = 20 ms vector, + = 30 ms vector, $\square$ = 40 ms vector. The scalar ECG demonstrates a transitional zone between lead V_1 and V_2 . The S-wave in lead V_2 is 2.8 mm, which is below the normal limit (5 mm). The ECG is coded 2.4 (chapter 9).

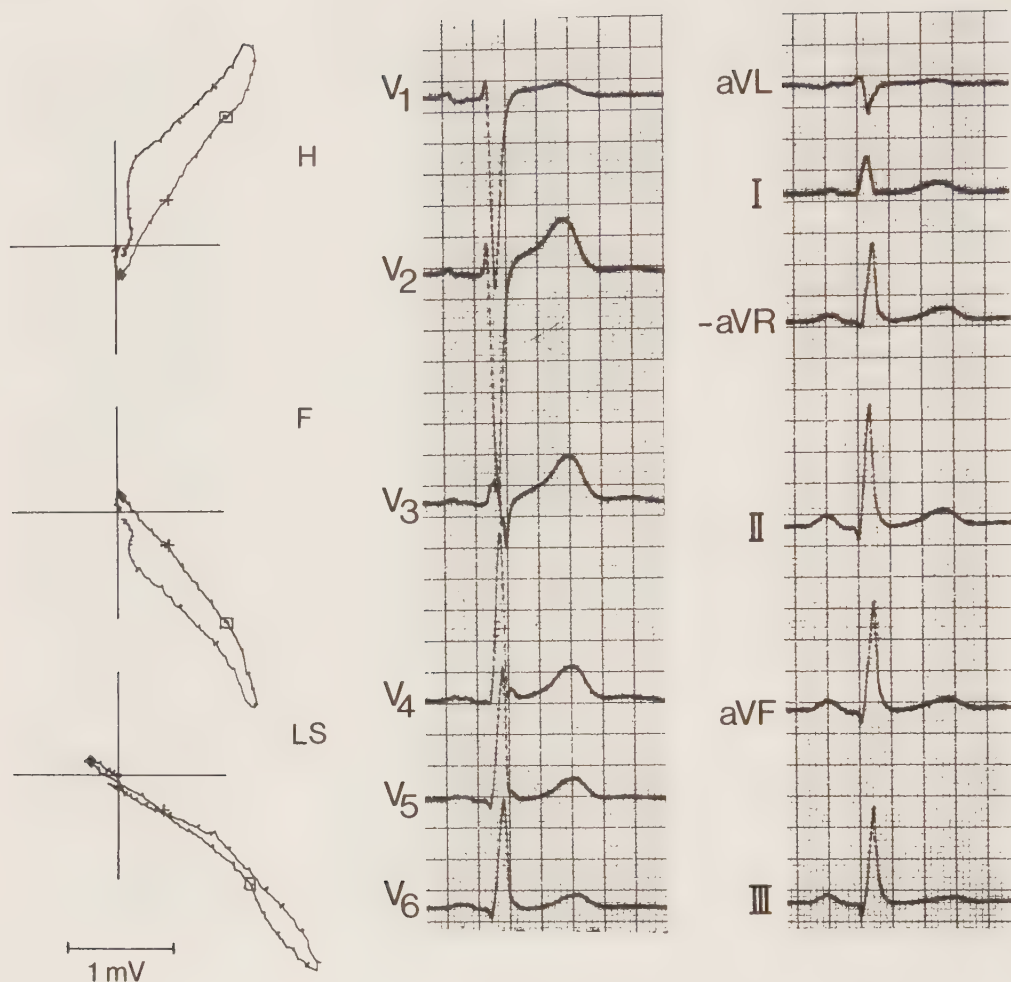


Fig. 8.8. Definitely abnormal QRS-loop on VCG of a man aged 32 years. A large maximal spatial vector of 2.92 mV was found in this VCG. Anterior defect and a figure-of-eight is seen in the left sagittal plane (LS). The VCG indicates hypertrophic cardiomyopathy or left ventricular hypertrophy with anterior infarction.

H = horizontal plane, F = frontal plane.

◇ = 20 ms vector, + = 30 ms vector, □ = 40 ms vector.

The scalar ECG demonstrates high amplitude S-waves in leads V_1 (30 mm) and V_2 (34.5 mm), which both exceeds the normal limits (20 resp. 32 mm). ECG-code 3.1 (chapter 9). The R-wave in lead V_3 gives an unexpected short and stout appearance, possibly reflecting the anterior defect seen in the VCG.

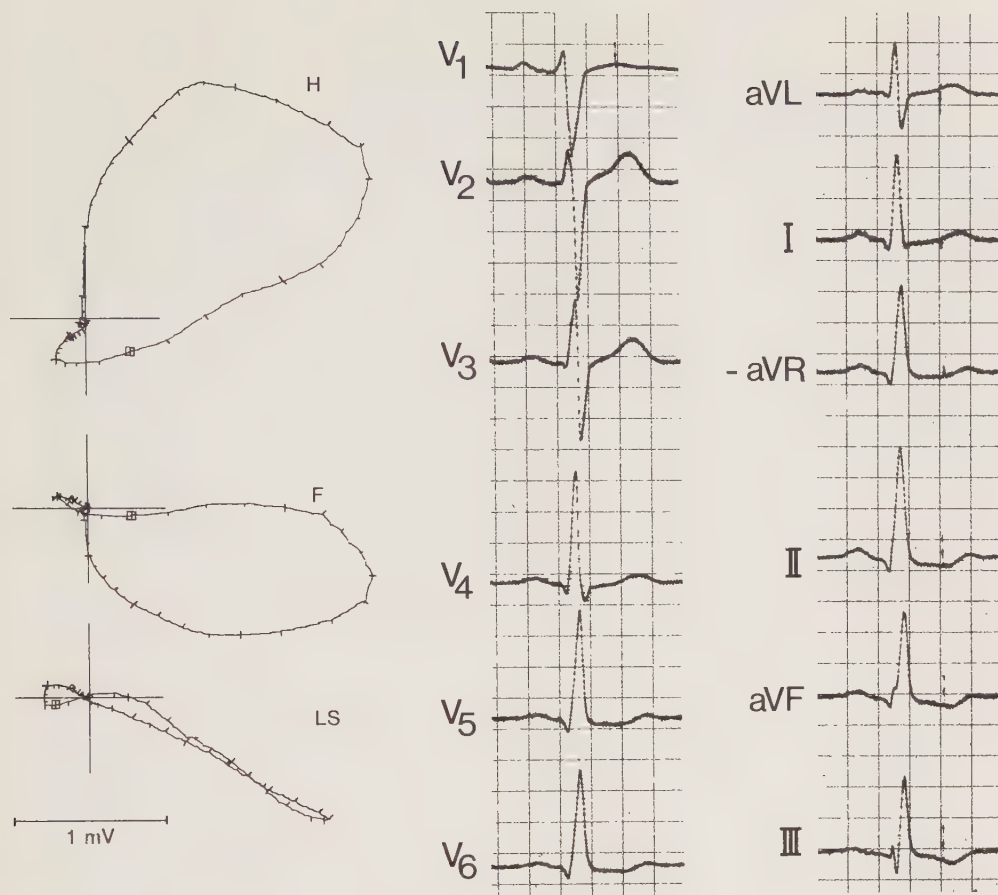


Fig. 8.9. Definitely abnormal QRS-loop on VCG of a man aged 31 years. Large defect anteriorly and inferiorly, suggesting low anterior or apical infarction.

H = horizontal plane, F = frontal plane, LS = left sagittal plane.

◇ = 20 ms vector, + = 30 ms vector, □ = 40 ms vector.

The scalar ECG demonstrate ischemic ST-segment changes (Punsar code I₄) in infero-lateral leads. The T-wave amplitude in lead aVF is negative, thereby exceeding the lower normal limit, which is +0.3 mm. Also in lead III the T-wave amplitude is deeper (-1.5 mm) than what is tolerated as normal (-1.0 mm). The notching of the QRS-complex in leads aVF and III are codable according to the new ECG-standard, proposed in chapter 9. ECG is coded 1.5, 4.1.3 and 5.2.1.

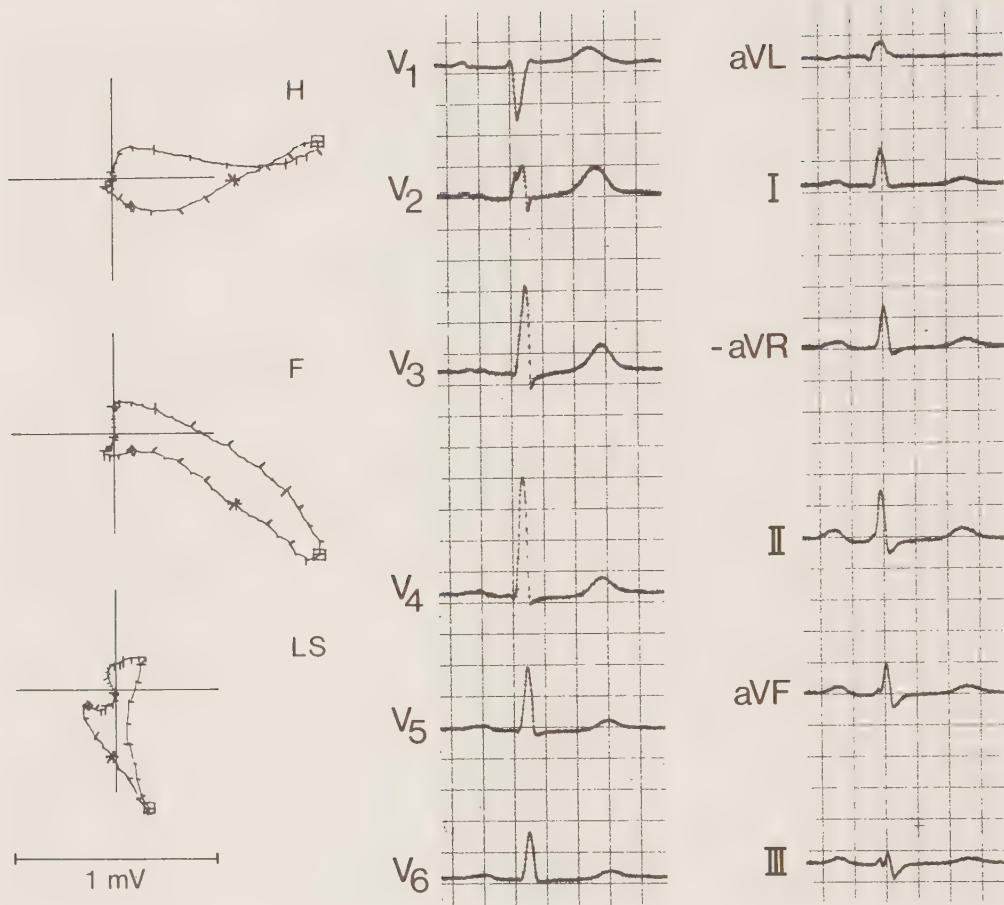


Fig. 8.10. Probably abnormal QRS-loop on VCG of a woman aged 48 years. Initial CW-rotation in the left sagittal plane (LS). Small posterior vectors and figure-of-eight in the horizontal plane (H) suggest posterior infarction.

F = frontal plane.

◇ = 20 ms vector, + = 30 ms vector, □ = 40 ms vector.

The scalar ECG demonstrates a precordial transitional zone between V_1 and V_2 . Also note the slowly ascending ST-segment in lead V_4 . The ECG is coded 2.4 and 4.2.2 (chapter 9).

9 AN ECG STANDARD FOR CLINICAL USE

Classification systems for ECG have proved valuable as a common language for the description of ECGs in population studies (Blackburn 1969). As yet, no classification system for clinical purpose has been suggested. With such a standard, also minor changes and other clinically used items (such as for instance notching) could be classified. A classification system for clinical use aims at a diagnosis of the patient while a classification system for population studies only aims at a uniform description of ECG-features. In population studies it is not necessary to consider, for instance, age and sex variations of the features. These and other variations may, in fact, be the mere objective of such studies. On the other hand, classification systems for clinical use must be designed to evaluate a certain feature in relation to the age and sex of the patient. Otherwise we would not know, if the feature is normal for the particular age and sex of that patient. The fact that a classification system for clinical use is meant to assist in the diagnosis of the patient, also implies that a code of such a system carries some pathological significance. An ECG standard for clinical use will be proposed here and it is based on the normal limits found in the present study. For ECGs, taken for some clinical reason, it was judged that these normal limits could be used to separate normal from abnormal ECGs. For population studies, however, this strategy would probably yield a rather high false positive rate.

The basic structure of this new standard is the same as in the Minnesota code (Rose and Blackburn 1968), although the individual items have been thoroughly modified to fit in the clinical context. Most of the ST-segment items are modifications of the Punsar code (Punsar et al 1968). The standard includes items for Q-wave, heart position, left ventricular hypertrophy (LVH), ST-segment and T-wave. As in the Minnesota code, the items and their subgroups are ordered so, that more serious codes are given lower numbers than less serious codes with exception for the LVH items (code 3), where the reverse order was found more practical. The basis for each of these items will be found in the respective section of this book. The standard does not include items on right ventricular hypertrophy, conduction defects, arrhythmias, atrial enlargement or low voltage.

All measurements of amplitudes are related to the PQ-segment and to a calibration of 1 mV = 10 mm.

The standard is applicable to leads aVL, I, -aVR, II, aVF, III and V₁-V₆ in the resting ECG with the subject in the supine position. The standard is applicable to Caucasian men and women, aged 21-60 years and probably also for older people, but it is important to note, that especially the heart position and hypertrophy items are not applicable to subjects younger than 21 years, as such subjects in general have higher amplitudes and different electrical positions than older subjects (Davignon et al 1970/80).

The standard considers variations with age and sex and heart position when necessary. However, minor influences from these factors have been overlooked in order to keep the standard as simple as possible.

Some comments on the hypertrophy items are warranted. Code A (chapter 5) has been slightly modified when included in the standard presented here. The modification implies that all ECGs, codable according to code A:1, are coded A:2 if left axis deviation (LAD) or ischemic ST-segments are present

in the ECG. By this it is possible to grade the probability of LVH, as has been done in code 3 in the standard, which will follow. Furthermore, the probability for LVH is strengthened if the terminal P-wave in lead $V_1 \geq 0.04$ s and at least -1 mm deep or the ventricular activation time (VAT) ≥ 0.05 s in lead V_5 or V_6 (Romhilt and Estes 1968). I therefore consider code 3.2 in the present standard as indicating possible LVH and code 3.3 as probable LVH.

The standard presented here is meant to be a guide to the clinician in the sense that it classifies an ECG as normal or abnormal. It is realised that opinions may differ about some items and that further validation is desirable. Nevertheless I would denote an ECG as abnormal if any of the codes, marked with an asterisk (*), was present and as probably abnormal if any of the codes without an asterisk was present.

Any classification system, which claims to be practical, implies simplification of the reality. The present standard is meant to be easy to use. It has therefore not been possible to consider variation with blood pressure or body build, two factors which have a significant influence on the appearance of the ECG. However, consideration to these factors is possible in computer programs for ECG-interpretation. Therefore, the influence of blood pressure and obesity on heart position and some amplitude measures have been analysed by means of multiple regression and reported in the appendix.

THE PROPOSED STANDARD FOR CLINICAL USE

This standard should not be used in the presence of CLBBB or pre-excitation

1. Q-wave and equivalent items

- | | | |
|-----|---|---|
| 1.1 | Any Q-wave duration > 0.03 s | Do not code QS |
| | If found in aVL: Code only if
$R_{aVL} \geq 3$ mm | |
| | If found in III: Code only if
Q-wave present
in aVF | |
| * | 1.1.1 Any Q-wave ≥ 0.04 s | |
| | 1.1.2 Any Q-wave > 0.03 s but
not 0.04 s | |
| * | 1.2 Any QS | Do not code aVL, III or V_1 |
| * | 1.3 Any Q-wave amplitude ≥ 0.4 mV and
Q/R-ratio $\geq 1/4$ | |
| * | 1.4 qrS or QS in V_1 and
Q-wave absent in V_6 | |
| 1.5 | Obviously notched (splintered)
R-waves in any two adjacent leads
of qR-configuration | Do not code terminal
notching, i.e. notching
at the base of the down-
stroke of the R-wave |
| | 1.5.1 In at least one of the two
leads notching must be
present in the Q-wave or on
the upstroke of the R-wave,
i.e. of either qqR-, qrR-
or qrsR-type | |
| | 1.5.2 Any precordial notching of
two adjacent qR-complexes | |
| 1.6 | R-wave amplitude ≤ 1 mm in all
three leads V_1 - V_3 | Do not code in the presence
of code 2.3 |

2. Heart position items

- | | |
|-----|--|
| 2.1 | Left axis deviation (LAD) |
| | R/S-ratio < 1 in aVF and
R/S-ratio > 1 in aVL |
| * | 2.1.1 LAD $< -30^\circ$ |
| | 2.1.2 LAD $< 0^\circ$ through -30° |

2.2 Right axis deviation (RAD)

R/S-ratio < 1 in lead I and

R/S-ratio > 1 in lead III

- * 2.2.1 RAD > 120°
- 2.2.2 RAD > 90° through 120°

- * 2.3 Precordial QRS-transition in V_5 or leftwards

- * 2.4 Precordial QRS-transition to the right of V_2

3. Left ventricular hypertrophy items

Do not use in presence of codes 1.1-1.4

3.1 High amplitudes (mm)

Do not code in presence of codes 2.1 or 4.1

	Women	Men ≥35 yrs	Men <35 yrs
$R_{aVL} \geq$	10	12	14
or $R_I \geq$	16	18	20
or SV_1 or $RV_6 \geq$	22	24	26
or $RV_5 \geq$	28	30	32

- * 3.2 If code 2.1 or 4.1 present:
As in 3.1 but reduce limits of 3.1 by 2 mm

- * 3.3 If code 3.1 or 3.2 present
and
terminal P-wave depression in V_1
at least -1 mm and 0.04 s
or
ventricular activation time at
least 0.05 s in V_5 - V_6

4. ST-segments itemsDo not code in presence
of CRBBB

- | | |
|--|---|
| <p>* 4.1 Horizontal or downward sloping ST-segments</p> <p>4.1.1 ST-junction depressed ≥ 1 mm</p> <p>4.1.2 ST-junction depressed ≥ 0.5 mm but not 1 mm</p> <p>4.1.3 ST-junction not depressed 0.5 mm but nadir of ST-segment ≥ 0.5 mm below the PQ-segment</p> | <p>Do not code III or V_1
Do not code leads with R/S-ratio < 1</p> |
| <p>* 4.2 Slowly ascending ST-segments
ST-junction depressed
ST-segment not traversing the base line before beginning of the T-wave</p> <p>4.2.1 ST-junction depressed ≥ 1 mm</p> <p>4.2.2 ST-junction depressed ≥ 0.5 mm but not 1 mm</p> | <p>Do not code III or V_1
Do not code leads with R/S-ratio < 1</p> <p>Do not code V_5-V_6 in women</p> |
| <p>4.3 Rapidly ascending ST-segments
ST-junction depressed
ST-segment traversing the base line before beginning of the T-wave</p> <p>4.3.1 ST-junction depressed ≥ 1 mm</p> <p>4.3.2 ST-junction depressed ≥ 0.5 mm but not 1 mm</p> | <p>Do not code III or V_1
Do not code leads with R/S-ratio < 1</p> <p>Do not code V_4</p> |
| <p>4.4 Minor ST-segment changes</p> <p>4.4.1 ST-junction zero or depressed but not 0.5 mm below PQ-level
Nadir of ST-segment zero or depressed less than 0.5 mm
The ST-segment may be horizontal, downward sloping or slowly ascending</p> | <p>Do not code in women
Do not code in aVL, aVF, III or V_1
Do not code leads with R/S-ratio < 1</p> |

- 4.4.2 ST-junction elevated but not more than 0.5 mm above the PQ-level
Nadir of ST-segment may be depressed but not 0.5 mm below PQ-level
The ST-segment may be horizontal or downward sloping, but not ascending

4.5 ST-segment elevation

ST-junction elevated more than

- 0.5 mm in any limb lead or V₅-V₆ in women
1 mm in any limb lead or V₅-V₆ in men
2 mm in any of V₁-V₄ in women
3 mm in any of V₁-V₄ in men

5 T-wave items

Do not code in presence of CRBBB

- * 5.1 Any T-wave inversion reaching 5 mm or more below PQ-segment

- * 5.2 Any T-wave, flat or inverted < 5 mm

Do not code III or V₁

- 5.2.1 Any T-wave inverted ≥ 1 mm but not 5 mm

If found in aVL:
code only if R/S > 1

- 5.2.2 Any T-wave inverted < 1 mm or flat

Do not code aVL

- 5.3 T-wave positive but T/R-ratio < 1/20

Do not code aVL, aVF, III or V₁

5.4 High amplitude T-waves

T-wave positive and at least

- 10 mm in any limb lead
12 mm in any chest lead in women
16 mm in any chest lead in men

SUMMARY

1. 472 randomly selected men and women from the city of Lund were examined for disease in the heart, lungs and for hypertension. 163 men and 194 women who had no symptom or sign of disease were accepted for the further study. The prevalence of various exclusion criterias, such as symptoms and signs of heart disease, lung disease and other diseases which may possibly affect the ECG are reported as well as the distribution of blood pressures in the sample.
2. A computer-averaged standard 12-lead ECG (leads aVL, I, -aVR, II, aVF, III, V₁-V₆) was recorded. All measurements of ECG-deflections have been made visually using a magnifying glass (6 times). ST-segments were classified according to the Punsar code by independent visual observers as well as by the computer.
3. The mean frontal QRS-axis shifted to the left with advancing age, but the shift was statistically significant only in men. In both men and women there was a leftward shift of the mean frontal QRS-axis with increased weight, increased chest circumference and increased obesity index. The normal range of axis was found to be 0° to 90° in men and +15° to 90° in women.

The problems concerning the definition of the electrical heart position is discussed. The concept of a Q-axis is introduced as an alternative way to indicate electrical heart position. There is a statistical significant relationship between the Q-axis and the QRS-axis in the frontal plane, although this relationship is not always apparent in the individual ECG. The presence or absence of a Q-wave in an individual lead was used to denote a lead as being a left ventricular lead or not. Using the Q-wave as a marker of heart position in the individual lead is more practical than to use the QRS-axis or the transitional zone.

4. Duration and amplitude of the Q-wave have been measured. The upper limit of normal duration exceeded 0.03 s in leads aVL and aVF in men but not in women.
5. The R-wave amplitudes proved to vary with age and heart position in men. In women variation of the R-wave amplitude was found with heart position but not with age. Upper and lower limits (2.5 % and 97.5 % percentiles) have therefore been determined, with due regard to these variations.
6. The S-wave amplitude was found to vary with age in lead V₁ and V₂ in men but not in women. Variation with heart position was found in men as well as in women.

7. New ECG-criteria for left ventricular hypertrophy have been proposed. The limits used are related to the upper limits of R- and S-waves found in this study. The criteria are easy to memorize and consider differences due to age and sex.
8. Validation of the new criteria for left ventricular hypertrophy was made in two unrelated samples with hypertensive men and women with aged matched controls. The results indicate, that the new code has a greater specificity than the Minnesota codes 3.1 and 3.2 (Rose 1968) and a better sensitivity than the point score system of Romhilt and Estes (1968). It is also possible that this new scalar code performs as good as one of the LVH-criteria derived from orthogonal ECG.
9. The amplitude of ST-junction was studied and a significant sex difference was found in most leads. The ST-junction amplitude in men is larger than in women in most leads.
10. The upward slope of the ST-segment, i.e. the inclination, decreases with age in men and women. There is a significant sex difference in all leads except lead aVL, men having steeper upward sloping ST-segments.
11. The prevalence of horizontal or downward sloping ST-segments with or without ST-junction depression was studied. The prevalence of horizontal or downward sloping ST-segments with elevation of the ST-junction was analysed and a code, I₆, is suggested as complement to the Punsar code system.

ST-segments, codable according to the Punsar code were significantly more common among women, due to a larger number of slowly ascending ST-segments. There was no difference between the sexes as regards ischemic ST-segment changes or rapidly ascending ST-segments.

12. The T-wave amplitude varied only slightly with heart position and age. The sex differences, on the other hand, were prominent in all leads except lead aVL, women having lower T-waves. Inverted T-waves were found to be normal in leads aVL, III and V₁.
13. A Frank lead vectorcardiogram (VCG) was recorded in all the subjects. Analysis of the VCGs indicates that 5 % of the subjects in the accepted sample may have silent heart disease. A detailed analysis of the VCGs will be reported in a separate study.
14. An ECG-standard for clinical use is proposed. The standard is based on normal values, found in the present study and also includes modified items from the Minnesota code and the Punsar code.

ACKNOWLEDGEMENT

The present study is the result of a team work. The following friends of mine have given freely of their energy and particular skills in order to help me to complete this study:

Professor *Håkan Westling*, Dean of the University of Lund and former head of the Department of Clinical Physiology

Professor *Björn Jonson*, head of the Department of Clinical Physiology

Elin Höglund, registered nurse and "chief coder"

Bodil Rickardsson Sjöberg, secretary of the project

Professor *Anders Gustafson*, head of the Department of Internal Medicine

Lisbeth Andersson
Kerstin Christensson
Lars Claesson
Jenny Holmquist
Gunilla Ivner
Agneta Jacobsson
Lisbet Jansson
Regina Johannesson
Majbritt Johansson
Ella Kjellman
Ingegärd Lindqvist
Kenneth Muir
Bodil Nilsson
Lisbeth Nilsson
Maria Nilsson
Olle Pahlm
Tore Persson
Ulla Rischell
Gun Sandström
Sven-Eric Svensson
Thomas Thulin
Olof Werner
Thomas White
Elisabeth Åström

I also extend warm thanks to the randomly selected inhabitants of this city for their co-operation in this study.

This study has been supported by the Swedish Medical Research Council, grant No. 14X-2872, the Swedish Association against Chest and Heart Diseases and the Medical Faculty of the University of Lund.

Generous contributions to the printing costs were given by SIEMENS-ELEMA, manufacturer of electronic equipment and by the following manufacturers of pharmaceutical specialities: CIBA-GEIGY Läkemedel AB, AB HÄSSELE, ICI-Pharma AB, MEDA AB and ROUSSEL Nordiska AB.

All individual data are available on request from the author or from the university library of the University of Lund.

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APPENDIX 1

ELECTRICAL HEART POSITION

Relationship between electrical heart position (expressed as the mean frontal QRS-axis, MFQRS-axis) and the frontal Q-axis and age, obesity index (OI), and diastolic blood pressure (DBP).

SD = residual standard deviation

t_{x1} = Students' t-value for x_1 , t_{x2} = Students' t-value for x_2

* = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$, NS = not significant

<u>MEN</u>	x_1	x_2	SD	t_{x1}	t_{x2}
MFQRS-axis	= 143 - 4.8 x OI - 0.65 x AGE		25.3	-3.7***	-3.4***
Frontal Q-axis	= 193 - 9.9 x OI - 0.73 x AGE		76.0	-2.5**	NS
MFQRS-axis	= 175 - 1.3 x DBP - 0.5 x AGE		24.8	-4.5***	-2.6**
Frontal Q-axis	= 218 - 2.0 x DBP - 0.6 x AGE		76.3	-2.3*	-1.0
MFQRS-axis	= 210 - 4.1 x OI - 1.3 x DBP		24.6	-3.2**	-4.7***
Frontal Q-axis	= 286 - 8.6 x OI - 1.7 x DBP		75.4	-2.2*	-2.0*
<u>WOMEN</u>					
MFQRS-axis	= 102 - 3.0 x OI - 0.17 x AGE		19.4	-3.8***	NS
Frontal Q-axis	= 89 - 3.0 x OI + 0.19 x AGE		77.3	NS	NS
MFQRS-axis	= 89 - 0.4 x DBP - 0.2 x AGE		20.0	-1.6	-1.2
Frontal Q-axis	= 135 - 1.2 x DBP + 0.4 x AGE		77.2	-1.4	0.6
MFQRS-axis	= 115 - 2.9 x OI - 0.26 x DBP		19.4	-3.6***	NS
Frontal Q-axis	= 148 - 2.0 x OI - 0.87 x DBP		77.2	NS	NS

APPENDIX 2

R-WAVE AMPLITUDE IN MEN

The influence of age, body build, diastolic blood pressure and heart position.

Table A. Mean values of R-wave amplitude (mm) and SEM in age groups in men. F-test has been applied and F-values are reported.

* = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$.

v_1 and v_2 = degrees of freedom.

1 mm = 0.1 mV.

Grand mean is the mean value for all men ($n=163$).

		LEADS	aVL	I	-aVR	II	aVF	III	V ₁	V ₂	V ₃	V ₄	V ₅	V ₆
A 21-30 years	n 66	M	2.1	5.5	8.8	13.1	11.0	9.1	4.0	8.0	10.6	17.4	17.0	12.9
		SEM	.19	.30	.27	.43	.49	.55	.23	.39	.60	.88	.71	.44
B 31-44 years	n 51	M	3.1	6.7	8.5	10.7	8.1	6.3	3.3	7.2	10.1	17.3	16.0	11.8
		SEM	.39	.45	.34	.54	.61	.58	.24	.52	.67	.94	.61	.43
C 45-60 years	n 46	M	3.3	6.3	7.3	8.6	6.1	4.6	2.5	5.7	9.9	16.7	15.7	10.8
		SEM	.39	.41	.31	.50	.58	.54	.18	.39	.60	.79	.74	.47
F-value v_1/v_2 2/160			4.5*	2.8	6.4**	22***	20***	17***	11***	7.0**	0.4	0.2	1.0	5.6**
Grand mean			2.8	6.1	8.3	11.1	8.7	7.0	3.4	7.1	10.2	17.2	16.3	11.9

(Appendix 2, contd.)

Table B. Variation of R-wave amplitude (mm) with age, body build, diastolic blood pressure and heart position in men (n=163). It should be noted that it has been possible to reduce the standard deviation substantially in most leads. The grand mean value and its SD are given for comparison. OI = obesity index, DBP = diastolic blood pressure, MFQRS-axis = mean frontal QRS-axis, TZ = precordial transitional zone, SD = standard deviation, r = correlation coefficient, t_{x1} = Students' t -value for x_1 , t_{x2} = Students' t -value for x_2 .

* = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$.

Values for the transitional zone are related to the lead in which the equiphasic QRS-complex is located. For instance, a value of 3 corresponds to a transitional zone in lead V_3 and a value of 3.5 to a transitional zone between leads V_3 and V_4 .

Lead	x_1	x_2	SD	r	t_{x1}	t_{x2}
<u>Lead aVL</u>						
R_{aVL} : Mean	2.8		2.37			
R_{aVL} =	$1.3 + 0.042 \times \text{AGE}$		2.33	0.20*		
R_{aVL} =	$-4.3 + 0.018 \times \text{AGE} + 0.1 \times \text{DBP}$		2.27		1.0	3.1**
R_{aVL} =	$-4.6 + 0.018 \times \text{AGE} + 0.5 \times \text{OI}$		2.20		1.1	4.5***
R_{aVL} =	$6.8 - 0.013 \times \text{AGE} - 0.06 \times \text{MFQRS-axis}$		1.62		-1.1	-13***
R_{aVL} =	$3.5 + 0.19 \times \text{OI} - 0.06 \times \text{MFQRS-axis}$		1.60		2.3*	-11.9***
<u>Lead I</u>						
R_I : Mean	6.1		2.85			
R_I =	$5.0 + 0.030 \times \text{AGE}$		2.84	0.12		
R_I =	$-0.9 + 0.004 \times \text{AGE} + 0.09 \times \text{DBP}$		2.79		0.2	2.7**
R_I =	$-4.2 - 0.008 \times \text{AGE} + 0.81 \times \text{OI}$		2.57		-0.4	6.1***
R_I =	$11.0 - 0.029 \times \text{AGE} - 0.07 \times \text{MFQRS-axis}$		2.23		-1.8	-10.1***
R_I =	$3.3 + 0.45 \times \text{OI} - 0.05 \times \text{MFQRS-axis}$		2.15		4.0***	-8.4***
<u>Lead -aVR</u>						
R_{-aVR} : Mean	8.3		2.30			
R_{-aVR} =	$10.3 - 0.054 \times \text{AGE}$		2.23	-0.26***		
R_{-aVR} =	$10.5 - 0.053 \times \text{AGE} - 0.004 \times \text{DBP}$		2.24		-3.1**	-0.1
R_{-aVR} =	$8.2 - 0.063 \times \text{AGE} + 0.19 \times \text{OI}$		2.22		-3.8***	1.6
R_{-aVR} =	$10.9 - 0.060 \times \text{AGE} - 0.007 \times \text{MFQRS-axis}$		2.23		-3.6***	-1.0
R_{-aVR} =	$7.2 + 0.066 \times \text{OI} + 0.003 \times \text{MFQRS-axis}$		2.32		0.5	0.5

(Appendix 2, contd.)

Lead II	x_1	x_2	SD	r	t_{x1}	t_{x2}
R_{II} : Mean 11.1			4.01			
R_{II} = 17.1 - 0.16 x AGE			3.58	-0.46***		
R_{II} = 25.6 - 0.13 x AGE - 0.13 x DBP			3.49		-4.7***	-3.1***
R_{II} = 22.6 - 0.14 x AGE - 0.48 x OI			3.51		-5.4***	-2.7**
R_{II} = 11.3 - 0.11 x AGE + 0.066 x MFQRS-axis			3.14		-4.5***	7.0***
R_{II} = 11.3 - 0.34 x OI + 0.074 x MFQRS-axis			3.29		-1.9	7.5***

Lead aVF

R_{aVF} : Mean 8.7			4.55			
R_{aVF} = 15.0 - 0.17 x AGE			4.14	-0.42***		
R_{aVF} = 26.8 - 0.12 x AGE - 0.17 x DBP			4.00		-3.9***	-3.8***
R_{aVF} = 27.1 - 0.12 x AGE - 11.1 x OI			3.81		-4.3***	-5.4***
R_{aVF} = 5.4 - 0.08 x AGE + 0.11 x MFQRS-axis			3.00		-3.4***	12.2***
R_{aVF} = 11.5 - 0.67 x OI + 0.11 x MFQRS-axis			2.92		-4.4***	12.0***

Lead III

R_{III} : Mean 7.0			4.57			
R_{III} = 12.8 - 0.16 x AGE			4.22	-0.39***		
R_{III} = 24.7 - 0.11 x AGE - 0.17 x DBP			4.06		-3.5***	-3.7***
R_{III} = 25.3 - 0.11 x AGE - 1.10 x OI			3.88		-3.8***	-5.5***
R_{III} = 2.9 - 0.06 x AGE + 0.11 x MFQRS-axis			3.01		-2.7**	12.5***
R_{III} = 9.7 - 0.67 x OI + 0.11 x MFQRS-axis			2.91		-4.4***	12.2***

(Appendix 2, contd.)

Lead V_1	x_1	x_2	SD	r	t_{x1}	t_{x2}
R_{V1} : Mean 3.4			1.75			
R_{V1} = 5.1 - 0.048 x AGE			1.67	-.31***		
R_{V1} = 7.5 - 0.052 x AGE - 0.73 x Tz			1.58		-4.6***	-4.4***
<u>Lead V_2</u>						
R_{V2} : Mean 7.1			3.34			
R_{V2} = 9.9 - 0.075 x AGE			3.24	-.25**		
R_{V2} = 16.7 - 0.086 x AGE - 2.10 x Tz			2.84		-4.3***	-7.0***
<u>Lead V_3</u>						
R_{V3} : Mean 10.2			4.64			
R_{V3} = 11.0 - 0.020 x AGE			4.65	-.05		
R_{V3} = 24.3 - 0.042 x AGE - 4.08 x Tz			3.51		-1.7	-11.1***
<u>Lead V_4</u>						
R_{V4} : Mean 17.2			6.51			
R_{V4} = 18.3 - 0.031 x AGE			6.52	-.05		
R_{V4} = 35.0 - 0.059 x AGE - 5.10 x Tz			5.29		-1.6	-9.2***
<u>Lead V_5</u>						
R_{V5} : Mean 16.3			5.17			
R_{V5} = 18.1 - 0.048 x AGE			5.15	-.10		
R_{V5} = 24.8 - 0.059 x AGE - 2.06 x Tz			4.93		-1.7	-4.0***
<u>Lead V_6</u>						
R_{V6} : Mean 11.9			3.42			
R_{V6} = 14.8 - 0.078 x AGE			3.31	-.26***		
R_{V6} = 18.2 - 0.084 x AGE - 1.02 x Tz			3.23		-3.7***	-3.0**

APPENDIX 3

R-WAVE AMPLITUDE IN WOMEN

Variation of R-wave amplitude (mm) with body build and heart position in women.

OI = obesity index, MFQRS-axis = mean frontal QRS-axis

Tz = precordial transitional zone

SD = standard deviation, r = correlation coefficient

t_{x1} = Students' t-value for x_1 , t_{x2} = Students' t-value for x_2

Grand Mean is the mean value for all the women (n=194).

Consideration has not been given to age and diastolic blood pressure in women because the influence was small. It should be noted that considerable reduction of the standard deviation has been obtained compared to the grand SD in many leads.

Values for the transitional zone are related to the lead in which the equi-phasic QRS-complex is located. For instance, a value of 3 corresponds to a transitional zone in lead V_3 and a value of 3.5 to a transitional zone between leads V_3 and V_4 .

	t_1	t_2	SD	t_{x1}	t_{x2}	Grand Mean $\pm$ SD
R_{aVL} = $1.6 + 0.22 \times OI - 0.04 \times \text{MFQRS-axis}$			1.28	4.1***	-8.4***	2.4 ± 1.62
R_I = $2.9 + 0.40 \times OI - 0.04 \times \text{MFQRS-axis}$			1.78	5.4***	-6.7***	5.8 ± 2.20
R_{-aVR} = $4.4 + 0.22 \times OI + 0.01 \times \text{MFQRS-axis}$			1.88	2.9**	1.1	7.9 ± 1.93
R_{II} = $5.0 + 0.08 \times OI + 0.08 \times \text{MFQRS-axis}$			2.59	0.8	8.1***	10.4 ± 3.05
R_{aVF} = $3.5 - 0.11 \times OI + 0.11 \times \text{MFQRS-axis}$			2.57	-1.1	11.6***	8.1 ± 3.47
R_{III} = $0.7 - 0.15 \times OI + 0.13 \times \text{MFQRS-axis}$			2.63	-1.4	13.3***	5.9 ± 3.78
R_{V_1} = $4.7 - 0.01 \times OI - 0.70 \times Tz$			1.29	-0.1	-4.2***	2.6 ± 1.34
R_{V_2} = $10.4 - 0.04 \times OI - 1.5 \times Tz$			2.41	-0.4	-4.7***	5.6 ± 2.54
R_{V_3} = $20.5 - 0.04 \times OI - 4.1 \times Tz$			3.00	-0.3	-10.4***	8.1 ± 3.75
R_{V_4} = $25.9 - 0.22 \times OI - 3.4 \times Tz$			3.77	-1.5	-7.0***	12.9 ± 4.22
R_{V_5} = $16.3 - 0.02 \times OI - 1.2 \times Tz$			3.68	-0.2	-2.6**	12.4 ± 3.73
R_{V_6} = $12.2 - 0.07 \times OI - 0.3 \times Tz$			2.84	-0.6	-0.9	10.3 ± 2.84

(Appendix 3, contd.)

		SD	r
R_{aVL}	$= 4.9 - 0.046 \times \text{MFQRS-axis}$	1.33	$-.57^{***}$
R_I	$= 8.8 - 0.055 \times \text{MFQRS-axis}$	1.91	$-.50^{***}$
R_{-aVR}	$= 7.7 + 0.002 \times \text{MFQRS-axis}$	1.94	.02
R_{II}	$= 6.2 + 0.077 \times \text{MFQRS-axis}$	2.64	$.51^{***}$
R_{aVF}	$= 1.7 + 0.11 \times \text{MFQRS-axis}$	2.60	$.67^{***}$
R_{III}	$= -1.5 + 0.13 \times \text{MFQRS-axis}$	2.64	$.72^{***}$
R_{V_1}	$= 4.6 - 0.70 \times Tz$	1.29	$-.29^{***}$
R_{V_2}	$= 9.9 - 1.47 \times Tz$	2.41	$-.32^{***}$
R_{V_3}	$= 20.0 - 4.05 \times Tz$	3.00	$-.60^{***}$
R_{V_4}	$= 22.8 - 3.38 \times Tz$	3.78	$-.45^{***}$
R_{V_5}	$= 16.0 - 1.23 \times Tz$	3.67	$-.18^{**}$
R_{V_6}	$= 11.3 - 0.33 \times Tz$	2.84	$-.07$

APPENDIX 4

S-WAVE AMPLITUDE

Table A. The S-wave amplitude (mm) in men and women with respect to presence or absence of a Q-wave in the lead. The number of S-waves are given in relation to the total number of ECGs, with or without Q-wave in the particular lead. Upper and lower limits as 2.5 % and 97.5 % percentiles. Lower limit is only reported in leads where an S-wave is mandatory. Students' test have been performed and statistical significance is reported. * = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$, NS = not significant. The S-wave amplitude in lead V_1 and V_2 are reported in section 4.3.1.

MEN (n=163)

q-wave present		aVL	I	-aVR	II	aVF	III	V_3	V_4	V_5	V_6
YES	n	55/86	79/105	81/123	74/116	54/103	46/95		58/64	106/132	103/163
	M	2.0	1.5	1.3	1.4	1.2	1.4		5.1	2.0	1.1
	SEM	.19	.10	.10	.12	.14	.20		.36	.15	.09
	SD	1.4	0.93	.87	1.1	1.0	1.4		2.7	1.5	.84
	Upper	5.6	3.3	3.8	4.3	4.7	6.8		11.6	7.4	3.8
NO	n	75/77	49/58	33/40	34/47	42/60	60/68	161/163	97/99	27/31	
	M	4.1	2.1	1.5	2.1	1.9	2.9	11.5	6.8	3.5	
	SEM	.25	.14	.15	.28	.23	.30	.38	.35	.41	
	SD	2.2	.96	.83	1.6	1.5	2.3	4.8	3.5	2.1	
	Upper Lower	9.7 0.6	4.6	3.9	6.2	5.5	9.9	24.2 3.1	15.5 1.2	9.6	
Student's test		***	***	NS	**	**	***		**	***	

WOMEN (n=194)

q-wave present		aVL	I	-aVR	II	aVF	III	V_3	V_4	V_5	V_6
YES	n	54/85	67/107	94/142	95/157	68/41	60/130		76/82	127/158	100/194
	M	1.2	1.0	1.0	1.2	1.1	1.0		3.1	1.4	0.8
	SEM	0.12	.08	.06	.08	.09	.10		.21	.07	.05
	SD	.88	.55	.56	.75	.77	.76		1.9	.78	.53
	Upper	3.8	2.3	2.1	3.0	3.3	3.3		7.8	3.1	2.1
NO	n	97/109	68/87	37/52	30/37	41/53	56/64	193/194	110/112	32/36	
	M	3.0	1.5	1.1	1.5	1.4	2.1	7.1	3.8	1.9	
	SEM	.20	.12	.11	.17	.15	.23	.23	.18	.26	
	SD	1.9	.99	.69	.92	.98	1.8	3.3	1.9	1.5	
	Upper Lower	7.5 1.3	4.2	2.9	3.4	4.1	9.2	13.4 0.7	7.3	4.5	
Student's test		***	***	NS	NS	NS	***		**	*	

(Appendix 4, contd.)

Table B. Variation of S-wave amplitude (mm) in lead V_1 and V_2 .

DBP = diastolic blood pressure, OI = obesity index, Tz, transitional zone in precordial leads. SD = standard deviation, r = correlation coefficient, t_{x1} = Students' t -value for x_1 , t_{x2} = Students' t -value for x_2 .

* = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$. Grand mean and SD are given for comparison and it should be noted that reduction of SD was obtained, especially in lead V_2 .

Values for the transitional zone are related to the lead in which the equi-phasic QRS-complex is located. For instance, a value of 3 corresponds to a transitional zone in lead V_3 and a value of 3.5 to a transitional zone between leads V_3 and V_4 .

MEN	x_1	x_2	SD	r	t_{x1}	t_{x2}
S_{V_1} : Mean 10.7			4.34			
$S_{V_1} = 14.5 - 0.10 \times \text{AGE}$			4.19	-.27***		
$S_{V_1} = 17.6 - 0.089 \times \text{AGE} - 0.047 \times \text{DBP}$			4.19		-2.7**	-1.0
$S_{V_1} = 17.9 - 0.088 \times \text{AGE} - 0.30 \times \text{OI}$			4.18		-2.8**	-1.4
$S_{V_1} = 11.0 - 0.096 \times \text{AGE} + 1.04 \times \text{Tz}$			4.15		-3.3***	2.1*
$S_{V_1} = 13.4 - 0.46 \times \text{OI} + 1.06 \times \text{Tz}$			4.22		-2.2*	2.2*
S_{V_2} : Mean 17.4			6.85			
$S_{V_2} = 26.4 - 0.25 \times \text{AGE}$			6.28	-.40***		
$S_{V_2} = 28.5 - 0.24 \times \text{AGE} - 0.030 \times \text{DBP}$			6.30		-4.9***	-.4
$S_{V_2} = 30.5 - 0.23 \times \text{AGE} - 0.36 \times \text{OI}$			6.28		-5.0***	-1.1
$S_{V_2} = 14.0 - 0.23 \times \text{AGE} + 3.80 \times \text{Tz}$			5.62		-5.7***	6.4***
$S_{V_2} = 14.6 - 0.72 \times \text{OI} + 3.95 \times \text{Tz}$			6.06		-2.4*	6.2***
WOMEN	x_1	x_2	SD	t_{x1}	t_{x2}	$M \pm SD$
$S_{V_1} = 13.3 - 0.29 \times \text{OI} + 0.25 \times \text{Tz}$			3.66	-2.0	.53	10.1 $\pm$ 3.68
$S_{V_2} = 10.9 - 0.73 \times \text{OI} + 4.59 \times \text{Tz}$			4.63	-4.0***	7.7***	14.3 $\pm$ 5.47***
	SD	r				
$S_{V_1} = 9.2 + 0.31 \times \text{Tz}$	3.69	.05				
$S_{V_2} = 0.6 + 4.72 \times \text{Tz}$	4.81	.48***				

APPENDIX 5

The ST-segment inclination (mV/s) in men and women in the 12 standard leads. Mean values and SEM are reported in three age groups, A, B and C. Significant age variations are indicated by the F-value. Grand mean is the mean value for all men (n=163).

* = $p < 0.05$, ** = $p < 0.02$, *** = $p < 0.001$, NS = not significant

MEN (n=163)

Lead \ Age	n		aVL	I	-aVR	II	aVF	III	V ₁	V ₂	V ₃	V ₄	V ₅	V ₆
A 21-30 years	66	M	0.2	0.5	0.6	0.6	0.4	0.1	0.6	2.0	2.2	1.5	0.8	0.5
		SEM	.023	.025	.025	.029	.028	.030	.059	.093	.088	.067	.043	.025
B 31-44 years	51	M	0.2	0.5	0.6	0.6	0.4	0.1	0.5	1.8	1.9	1.4	0.9	0.6
		SEM	.028	.033	.030	.045	.043	.045	.056	.10	.082	.065	.053	.035
C 45-61 years	46	M	0.1	0.3	0.4	0.4	0.3	0.1	0.4	1.3	1.4	1.0	0.6	0.3
		SEM	.027	.030	.029	.032	.029	.035	.043	.080	.075	.068	.057	.043
F-value v ₁ /v ₂ = 2/160			**	***	***	***	***		*	***	***	***	***	***
			6.4	20.1	19.4	13.5	9.5	NS	3.35	14.6	22.5	14.7	8.6	19.5
Grand Mean			0.2	0.5	0.5	0.6	0.3	0.1	0.5	1.7	1.9	1.3	0.8	0.5

WOMEN (n=194)

Age \ Lead	n		aVL	I	-aVR	II	aVF	III	V ₁	V ₂	V ₃	V ₄	V ₅	V ₆
A 21-30 years	83	M	0.2	0.4	0.4	0.4	0.2	0	0.1	0.9	0.9	0.7	0.5	0.3
		SEM	.019	.023	.021	.023	.020	.023	.029	.053	.042	.031	.026	.022
B 31-44 years	63	M	0.1	0.3	0.3	0.3	0.2	0	0.2	0.9	0.9	0.6	0.4	0.3
		SEM	.015	.018	.021	.027	.025	.024	.029	.057	.042	.034	.028	.023
C 45-61 years	48	M	0.1	0.2	0.3	0.3	0.2	0	0.3	0.9	0.9	0.6	0.4	0.2
		SEM	.016	.020	.020	.024	.022	.020	.039	.053	.060	.052	.027	.022
F-value v ₁ /v ₂ = 2/191			***	***	***	***			***			**	***	***
			27.1	21.6	18.8	13.7	NS	NS	10.1	NS	NS	4.6	8.5	7.1
Grand Mean			0.2	0.3	0.4	0.4	0.2	0.01	0.2	0.9	0.9	0.6	0.4	0.3

APPENDIX 6

The peak to peak QRS-deflection amplitude (mm) in men and women in the 12 standard leads. Subjects are divided into two age groups (Y and O). Age differences have been tested with Students' t-test and the t-values are reported. 1 mm = 0.1 mV.

SD = standard deviation. UL = upper limit as the 97.5 % percentile.

LL = lower limit as the 2.5 % percentile.

* = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$

MEN n=163		aVL	I	-aVR	II	aVF	III	V ₁	V ₂	V ₃	V ₄	V ₅	V ₆
Y age 21-34 years n=89	Mean	5.8	7.8	10.3	14.3	11.8	10.4	15.5	27.5	22.7	23.8	19.4	14.2
	SD	2.6	3.0	2.6	3.8	4.5	4.7	5.3	7.5	7.0	7.6	5.3	3.6
	UL	14.0	16.7	16.6	22.5	20.3	20.4	28.5	44.1	39.3	42.7	35.0	22.7
	LL	1.5	3.6	5.5	6.3	2.7	2.9	7.0	12.5	8.8	11.5	9.8	7.8
O age 35-60 years n=74	Mean	5.4	7.9	8.7	10.6	8.0	7.3	12.3	20.8	20.4	22.6	18.1	12.4
	SD	2.7	3.0	2.1	3.8	4.1	3.5	4.3	5.7	4.5	5.5	5.3	3.5
	UL	11.8	15.4	12.7	17.6	16.8	17.3	23.6	32.6	32.3	33.1	31.8	23.1
	LL	1.2	3.5	5.1	4.2	1.5	2.0	5.1	8.9	11.0	11.4	9.3	5.5
t-value		1.0	0.2	4.3***	6.2***	5.6***	4.7***	4.2***	6.3***	2.4*	1.1	1.6	3.2**

WOMEN n=194		aVL	I	-aVR	II	aVF	III	V ₁	V ₂	V ₃	V ₄	V ₅	V ₆
Y age 21-34 years n=108	Mean	4.4	6.9	9.0	12.1	9.7	7.8	12.5	20.5	14.9	15.9	13.9	11.5
	SD	1.9	2.2	2.0	3.1	3.5	3.9	4.5	6.4	4.5	4.0	3.8	3.1
	UL	8.4	11.1	13.7	20.1	18.1	16.3	23.8	36.1	24.9	24.3	21.8	19.2
	LL	1.4	3.0	5.0	6.4	3.6	2.1	4.7	9.1	7.1	8.2	7.4	6.5
O age 35-60 years n=86	Mean	4.4	7.0	8.7	11.2	8.7	6.8	12.7	19.3	15.6	17.3	14.2	11.0
	SD	2.2	2.5	1.9	3.1	3.8	3.9	3.9	6.2	4.9	4.6	3.8	2.8
	UL	11.8	11.6	13.2	19.1	17.8	17.2	20.8	38.2	28.5	29.0	25.0	17.6
	LL	1.6	2.4	4.8	5.3	2.5	1.9	6.0	7.3	7.3	8.7	6.4	6.4
t-value		0	-0.3	1.1	2.0*	1.9	1.8	-0.3	1.3	-1.0	-2.3*	-0.5	1.2

APPENDIX 7

NORMAL VALUES OF INTERVALS

This book has not dealt with intervals except Q-wave duration, although the PQ-, QRS- and QT-duration have been studied as well. During the last minute preparation of this book it was, however, decided to publish also the normal values of these intervals. The PQ-intervals have been measured from the beginning of the P-wave to the beginning of the QRS-complex in lead II and the QRS-duration has been measured in lead V₁. The QT-interval (from the beginning of QRS to the end of T) was corrected for heart rate (QT_c) according to the modified formula of Bazett (Kissin 1948), which is $QT_c = QT/\sqrt{60/HR}$. All measurements are made visually to the nearest 0.002 s. The normal values of PQ- and QRS-duration conform well with the values used in the Minnesota codes 6 and 7, although sex differences with shorter intervals in women were seen. Measurements in seconds.

Differences tested with Students' test. *** = $p < 0.001$, NS = not significant
UL = 97.5 % percentile, LL = 2.5 % percentile

		Men (n=163)	Difference	Women (n=194)
PQ- duration (lead II)	M	0.16		0.15
	SD	0.021		0.021
	UL	0.22	***	0.20
	LL	0.12		0.11
QRS- duration (lead V ₁)	M	0.090		0.081
	SD	0.0094		0.0083
	UL	0.11	***	0.10
	LL	0.07		0.06
QT _c (lead II)	M	0.416		0.422
	SD	0.036		0.026
	UL	0.488	NS	0.462
	LL	0.367		0.378

Normal limits of QT_c in lead II if the heart rate (HR) is 60-100 per/min

	Men (n=130)	Women (n=175)
UL	0.476	0.462
LL	0.372	0.381

ISBN 91-7222-714-1